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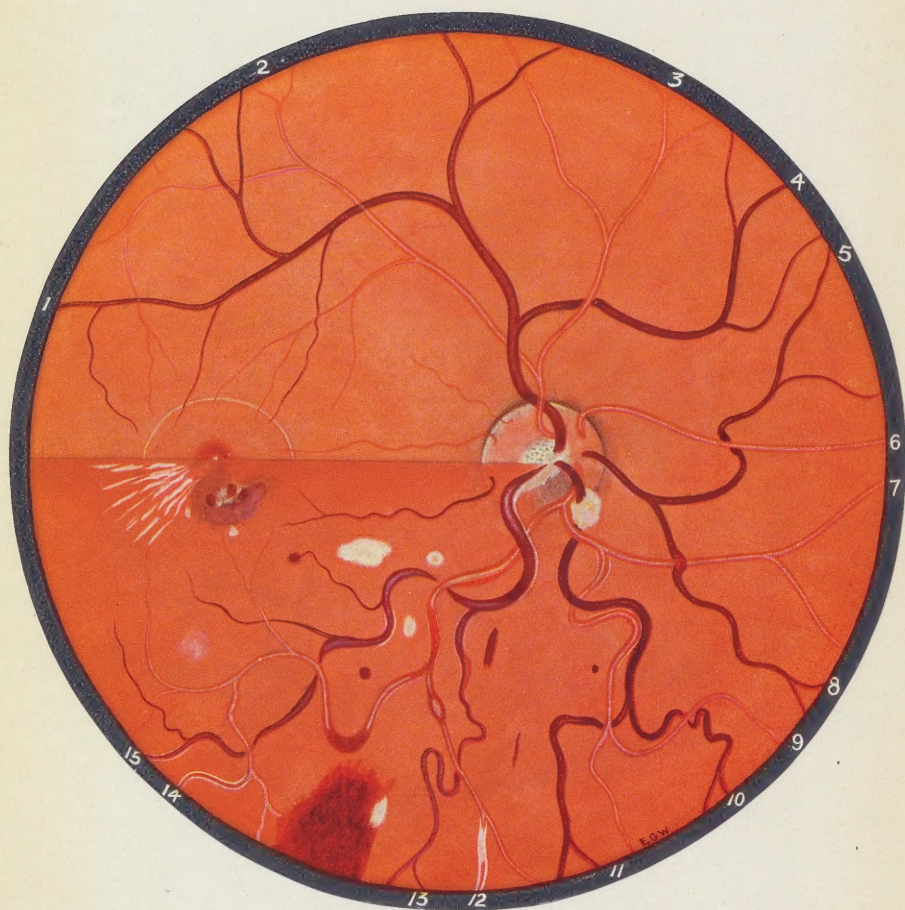
Section

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W. D. S. G. O.

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BLOOD PRESSURE
IN
OCULAR WORK



A COMPOSITE FUNDUS

Illustrating Progressively, as the figures read, some of the changes which take place as the result of Nephritic, Diabetic, Arteriosclerotic, Luetic and other diseases mentioned in this volume.

[“Blood Pressure in Ocular Work.” Wiseman.]

COMPOSITE FUNDUS

Showing progressively the changes which take place in the more common diseases.

To be read from the upper left quadrant to the right.

1. A normal vein, displaying the usual sinuosity. 2. A normal artery, somewhat straighter than the vein. The latter can be seen through the former, showing that the walls of the artery are transparent. 3. Artery with walls no longer transparent, as underlying vein is concealed. 4. Underlying vein depressed by a stiffened artery. 5. Underlying vein dimpled by rigid artery, causing ampulliform dilatation. 6 and 7. Silver-wire arteries. 8. Vein assuming band-like formation as it passes over rigid artery, being pressed flat against the cylindrical vessel by the intraocular tension. 9. Artery greatly increased in tortuosity, displaying prominent light streak and pushing vein aside with one loop. 10. Vein displaying irregularity in calibre; markedly tortuous and swollen as it approaches artery. Further along it is thrust aside by the stiffened artery. 11. Temporal branch of the same vein, showing vessel dilated as it approaches overlying artery, the latter also depressing it very noticeably. 12. Nasal twig of temporal artery displaying marked perivasculitis. The blood column at one point is so completely ensheathed in opaque walls that it cannot be seen. Later it swells into a small aneurysm. 13. Dimpled and tortuous vein with its blood column interrupted by the overlying artery, as the reduced calibre of the vein beyond the artery would indicate. The same vessel beyond the artery has an extravasation of blood into the adjacent tissue which constitutes a linear hemorrhage. Just beyond the hemorrhage, the vessel has a faint light streak which is characteristic of semi-advanced cases. 14. Artery showing, progressively, marked perivasculitis, depression of vein, prominent light streak, "pinched" vein, aneurysmal formation at bend of vessel, irregular calibre, loss of light streak, irregular color, linear extravasation-hemorrhage. 15. Veins displaying, progressively, perivasculitis, depressed and concealed condition, irregular calibre, marked tortuosity, light streak, tapering calibre at the disk where the vessel is well-lined with exudate. A small branch terminates in a slight hemorrhage. The condition has been observed in the terminal stages of diseases displaying a high blood-pressure.

The upper half of the macula is normal. The lower right hand quadrant displays a condition frequently seen, in varying degrees, in cases accompanied by a high or moderately high blood-pressure. The lower left quadrant shows the stellate picture of Bright's disease.

The upper half of the nerve is normal, showing a healthy flushed condition, the lamina cribosa, and the choroidal and scleral rings. The lower right quadrant illustrates the dull gray condition observed in some cases of hypertension. The condition is probably atrophic. The lower left quadrant illustrates the highly inflamed condition frequently seen. The margins of the nerve are practically obliterated. The blood-pressure depends upon the disease causing the hyperemia.

It is difficult, if not impossible, to portray on a plate of this kind the numberless variations that occur in the general appearance of the fundus, but the gradual increase in the intensity of the red reflex serves to illustrate the expected changes that may take place concomitant with the advance of the disease. The upper half may be taken as relatively normal. The blood-pressure associated with it is usually normal. The lower displays the increased hyperemia, the black spots of pigment, patches of exudate and round and striate hemorrhages. The large hemorrhage is characteristic of the general fundus picture in thrombosis of the central vein. In such cases the first ophthalmoscopic view of the fundus shows it to resemble, more or less completely, a sheet of blood covering the entire field. When only a branch vessel is involved, only the sector supplied by it is affected, there sometimes being, under such circumstances, a roughly shaped triangular area well covered with individual hemorrhages. The blood-pressure associated with such conditions is usually high. (WISEMAN).

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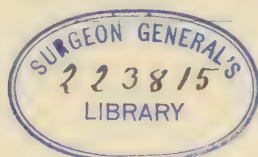
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A TREATISE
ON
BLOOD PRESSURE
IN
OCULAR WORK

WITH
SPECIAL REFERENCE TO FACTORS
OF
INTEREST TO REFRACTIONISTS

BY
EUGENE G. WISEMAN

ILLUSTRATED WITH 19 ENGRAVINGS



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TO MY WIFE,
*Whose Idealism Has Inspired
This Effort to Serve Humanity,*

and

TO MY MOTHER,
*Whose Teaching, by Precept and
Example, of the Value of Patience,
Has Made Its Inscription Possible,
This Book Is Affectionately
Dedicated.*

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INTRODUCTION

The particular purpose of this volume is to acquaint the optometric profession with a science hitherto unemployed by it, but one which vitally concerns its future welfare. Just so long as optometrists confine themselves rigidly to the ascertainment of the refractive and muscular status of the eye and neglect those factors which arouse symptoms simulative of ocular strain, just so long will optometry fail to rise to the height to which the importance of the work justly entitles it, and so long will its individual practitioners shine, at best, with the dim light of quasi-professionalism.

Differentiation between symptoms caused by local functional abnormalities remediable with lenses, and symptoms aroused by morbid pathological processes situated locally or remotely and remediable by medical or surgical measures, is the *sine qua non* of the future progress of the profession. That is actually what the public seeks and, to a notable measure, it is something the public is not able to obtain, *in any profession now extant*. The opportunity presented to the practitioners of optometry is obvious and unique.

The author is well aware of the fact that his advocacy of the use of this science by optometrists will meet with bitter opposition—perhaps with personal attacks. Those without the profession who have so strongly fought against the elevation of optometric standards will natu-

rally endeavor to discourage a step that will so greatly widen the sphere of the optometrist's usefulness, while those within the profession who are laggards in the intellectual race for supremacy, and who do not wish to be outstripped in the future, will naturally seek to hold back those who desire to ascend to newer, broader and more useful spheres of action.

His experience with the science of sphygmomanometry has been such that he knows those who seek to oppose its amalgamation with the science of optometry are actuated either by a spirit of ill-will toward the profession or by ignorance of the true merits of the former science. There can be no actual justification for such opposition, as there is no substantial foundation upon which to base the premises.

The former type of opponent can be ignored; the latter can be taught. In reply to the latter, it can be said that long since ophthalmoscopy has been deemed a very essential adjunct to optometry, yet it is undeniably true that the ophthalmoscope merely reveals information concerning destructive tissue-changes that have already taken place, and which, in the great majority of instances, cannot be rectified wholly, and seldom in part. *If it is important to detect such diseases with the ophthalmoscope after the destruction has taken place, is it not far more important to detect symptoms of the fundamental causes of such disturbances in time to avert the impending disaster?* The answer is so obviously in the affirmative that the strongest negation can be only inconsequential.

Again, if it is desirable to relieve the symptoms of eye-strain by means of lenses, is it not imperative for the operator to know whether the symptoms complained of are aroused by such a condition or by something entirely

foreign to optical or muscular anomalies? Here, too, the answer can be only in the affirmative. Then it follows that any reasonable means that can be utilized for the purpose of promoting competent service in these two respects should be employed by the optometrist.

The author is cognizant of nothing that so completely fulfills the requirements as does sphygmomanometry. The wide application of the science, the comparative simplicity of the principles involved (as far as its use by the optometrist is concerned), the celerity with which information may be obtained, and the admirable adaptation of the instrument employed to the optometrist's armamentarium render it a peculiarly appropriate adjunct and one the writer is convinced will form the nucleus of a better understanding and a higher appreciation of our work.

It is not the author's claim to have advanced much that is original in this volume. It is a matter of course that the bulk of the material presented, particularly that relating to pathological processes, has been gathered from medical literature, the indebtedness being acknowledged in most instances, particularly where *verbatim* quotations are made. Especially has Knies' excellent work, "The Eye in General Diseases," been quoted from extensively, as it is the most complete work of its kind that the author has been able to obtain. The new American Encyclopedia of Ophthalmology also has been freely used, with the generous permission of the Editor, as it presumably constitutes the last word in ophthalmic science. It occasions great regret that the set is not yet complete, as subsequent volumes undoubtedly will contain much that is of value.

Notwithstanding the author's non-assumption of undue credit, he believes it to be true that this volume

presents the relationship between the blood-pressure and the optometric phase of ocular work not only in a partially new light, but in a more just proportion. In other words, the true significance of the associated phenomena is more clearly presented than in any other work of which he is aware.

While it is a fact that all recent ophthalmological treatises of importance touch upon the pathological influences of abnormal blood-pressure and its causes upon the ocular economy, reference to the optical side of the question is only passing, if made at all. It is impossible, then, to obtain from them an adequate understanding of this vastly important subject, a subject which directly concerns at least 22.5 per cent, and possibly 47.5 per cent, of all cases which appeal to the optometrist—and, perhaps, to the ophthalmologist—for relief.

This fact has rendered it imperative that the seeker after knowledge concerning this subject wade through an enormous mass of medical and allied literature in order to obtain a comprehensive understanding of the various phenomena involved. With this as a basis, the connection proper between blood-pressure abnormalities and their causes, refractive and muscular anomalies, and associated symptoms may be studied and the desired result achieved—if the student does not weary of the pursuit long before he has reached his goal.

That others have been reluctant so to do is very evident from the present need for a volume of this kind. Therefore, in order to simplify this study, eliminate superfluous matter and present the subject in a logical and condensed form that those less patient of detail and more pressed for time may grasp it with reasonable celerity, the writer has undertaken to cull the essentials

from the material at hand and present them in the following chapters. It is hoped that his efforts will meet with approbation.

The final chapter, "Sphygmomanometry in Optometric Practice," is believed to be almost entirely original, both in its manner of presentation and in the subject matter contained. Notable exception is made only in regard to Group E, almost in its entirety, and to certain cases in Group D, especially in Class 3.

The chapter upon "Sphygmomanometry" has been carefully written and the subject presented in more or less detail, that the student may be well grounded in this subject. Whatever originality it may possess relates to the interpretation of the five phase-phenomena encountered in auscultation.

All throughout, medical words and phrases have been utilized wherever advisable, as the writer deems it highly desirable that optometrists, in taking up the study and application of blood-pressure, be prepared to discuss it in medical language with those members of the medical profession with whom they come into contact. This will not only enable them to avoid many embarrassing situations, but it will give them a more intelligent understanding of the subject. The appended glossary simplifies matters in this respect.

No apology is needed for the bald presentation of luetic disease. An apparent prudishness in this respect in the past has effectually prevented the dissemination, among optometrists at least, of knowledge concerning this highly important subject.

The author wishes to take advantage of this opportunity to acknowledge his indebtedness to William H.

Hammond, of Lockport, N. Y.. for inspiration offered, perhaps all-unconsciously, by him during the several years of friendship and mutual understanding that have existed between them.

Her patience in transcribing notes, and faithfulness in sticking to a not-very-pleasant task, renders the author very greatly indebted to his secretary, Miss Lydia Hennig, without whose aid this work might have been postponed indefinitely.

EUGENE G. WISEMAN.

BUFFALO, New York.
December 1915.

BLOOD PRESSURE IN OCULAR WORK

SECTION ONE

Fundamentals of Blood-Pressure Determinations

CHAPTER I

GENERAL CONSIDERATIONS

The axiomatic expression "an ounce of prevention is worth a pound of cure" is as true today as when first uttered. It is never more appropriate than when applied to the ills "that flesh is heir to."

The Hibernicism—men grow old too young—has its justification in the fact that the ounce is not applied until the pound is required. If all men followed the rules of right living as now known, it is a safe prediction that the length of the average life would be amazingly increased.

This assertion is warranted not only by casual observation, but by proof as conclusive as it is possible to obtain. By a coincidence, this proof came to the author's hand at the very time, almost to a day, it was most needed, in the form of an address delivered by Mr. Arthur Hunter, Actuary of the New York Life Insurance Co., before

The Association of Life Insurance Presidents, entitled "Can Insurance Experience Be Applied to Lengthen Life?"

Mr. Hunter's laconic answer to his own question is "It can," and he endeavors to substantiate it by presenting an analysis of a scientific investigation of two million insured lives.

It is unnecessary to give in detail the method by which he arrives at his results or, indeed, the results themselves *in toto*, but the appended notes on page 238 pertinently express his conclusions and contain, in addition, comments thereon. (The reader is urged to carefully go over this matter, as it certainly is germane to the subject.)

Even a casual analysis of these notes justifies the conclusion that since human nature is ever the same, there will always be with us those who are paying, or will pay, the consequences of indiscretion. It then behooves those who are engaged in any form of therapeutical science to be at least partially acquainted with the symptoms of such consequences, especially when they relate more or less directly to that phase of the science in which the practitioner is engaged.

For this reason this chapter is presented to optometrists. While the facts it contains may seem, at first glance, to be extraneous to optometric practice, careful consideration of them will indicate that, on the contrary, they are at the very basis of that phase of our work which has to do with preventive measures. That this phase is becoming of ever-increasing importance, the prefatory remarks have pointed out.

When it becomes clear to the optometrist that certain diseases predominate in vital statistics; that these diseases predominate among the causes of ocular disease in

general; that in particular the majority of the visual disturbances of a pathologic nature which he encounters in his office practice are due to such diseases, and that a high percentage of his failures are due to them, he must, perforce, realize the importance of having a passing acquaintance with them, and with methods of detecting them. The sequence of facts constitutes a pyramid, the apex of which has a personal application and should serve as a prod to stimulate a proper appreciation of the need of eliminating the causative factors of disease at their point of orientation.

In considering this subject, it is appropriate to give precedence to the basic factor—general pathology—and that can best be done by referring to statistics which are incontrovertible.

An investigation of the Sixty-third Annual Statement of the Massachusetts Mutual Life Insurance Co. reveals the information that of 968 deaths in 1914, the major causes were as follows:

SPECIFIC

Diseases of kidneys.....	108
Apoplexy.....	99
Pneumonia.....	61
Tuberculosis (Pulmonary).....	63
Cancer and other tumors.....	80

GENERAL

Circulatory { Heart.....	148
System { Arteriosclerosis.....	28
Digestive system.....	87
Nervous system (other than apoplexy).....	31

The New York Life Insurance Company's mortality table is classified somewhat differently, but in effect it is practically the same as that of the Massachusetts Mutual Life Insurance Co. The number of deaths recorded is 8,848, and the table is given herewith:

BLOOD PRESSURE IN OCULAR WORK

SPECIFIC

Diseases of kidneys.....	894
Apoplexy.....	624
Pneumonia.....	704
Tuberculosis (Pulmonary).....	907
Cancer and other tumors.....	708

GENERAL

Circulatory {Heart.....	1072
System {Arteriosclerosis.....	319
Digestive system.....	588
Nervous system (other than apoplexy).....	395

Lest an insurance table seem too selective, and not applicable generally, there is also given a list of the major causes of death as issued by the Buffalo Department of Health. The table applies to the year 1912, and is presumed to be a fair example of other years.

From the total number of deaths—6,527—there have been subtracted 1,904, that being the number of children under five who died from various causes. Since children under that age are rarely, if ever, afflicted with the diseases encountered in optometric work that are presently to receive consideration, it is deemed but fair that a distinction should be made. Likewise, children over five years of age are included because they have progressed beyond the diseases of infancy which cause such an enormous mortality. It is understood, then, that the following table applies to all over five years and represents the major causes of death in a total of 4,623:

SPECIFIC

Diseases of kidneys.....	460
Apoplexy.....	280
Pneumonia.....	332
Tuberculosis (Pulmonary).....	525
Cancer and other tumors.....	305

GENERAL

Circulatory {Heart.....	576
System {Arteriosclerosis.....	246
Digestive system.....	376
Nervous system (other than apoplexy).....	228

Analysis of these reports results in the following table which gives in percentages the ratio between the total number of deaths and the number due to each disease:

	Mass: Mut: Life	New York Life	Buf. Dept. Health	General Average
Diseases of kidneys.....	11.1	10.1	9.9	10.3
Apoplexy.....	10.2	7.05	6.05	7.7
Pneumonia.....	6.3	7.9	7.3	7.1
Tuberculosis (Pulmonary).....	6.5	10.2	11.1	9.2
Cancer and other tumors.....	8.2	8.0	6.5	7.5
Circulatory {Heart.....	15.4	12.1	12.4	13.3
System {Arteriosclerosis.....	2.8	3.6	5.3	3.9
Digestive system.....	8.9	6.6	8.1	7.8
Nervous system (other than apoplexy).....	3.2	4.4	4.9	4.1
Percentage of total deaths.....				67.9

Fig. 1

Table Showing the Percentage of Deaths due to the Several Diseases mentioned.

HEART DISEASES

The largest single factor disclosed by the above table is heart disease. Just why diseases of that organ should predominate, it is difficult for the non-medical practitioner to understand, unless they are largely reflex affections from diseases of other organs, the kidneys, for instance. Indeed, of the effects of kidney disease upon the body at large, Bradford and Owen assert that its effect upon the cardiovascular system is second only to its effect upon the balance of the entire system. It follows, apparently as a matter of course, that its direct effect upon the arteries produces an indirect effect upon the heart, so that the two conditions are more or less interdependent and may often be considered virtually as one.

It should be remembered that in all of these reports the *actual* cause of death is given and not that circumstance which led to the final condition. For example, a high percentage of deaths are attributed to APOPLEXY,

by which is chiefly meant a cerebral hemorrhage. Now it is clearly evident, from the chapter upon arteries, that the bursting of an artery is improbable if the vessel is not diseased. It follows, then, that apoplexy, classified sometimes under diseases of the nervous system, is in reality due *directly* to diseases of the vascular system, and very often *indirectly* to diseases of the renal system.

It is the same with heart disease. The chain of circumstances involved may be ingestion of improper food materials, then insufficient or improper elimination by the renal system, a resultant effect upon the arteries, which become hardened and perhaps have their lumen narrowed, and a consequent over-exertion of the heart in order to overcome the increased arterial resistance to the blood flow. Finally, the heart cannot sustain the effort and it fails. The cause is classified as heart failure, and it *is* such, but back of all is kidney and arterial disease, superinduced by improper eating and drinking. Of course, this picture is not truly applicable to every case, for syphilitic and other diseases may be the inciting causes, but it serves to illustrate the point.

Owing to these facts, it is believed that the apparent predominance of heart disease is deceptive.

Be that as it may, the direct or indirect effect of heart disease, *per se*, upon the ocular economy appears to be so insecurely established that it need be little considered here. Outside of embolism of the central artery of the retina, which might result by there becoming detached, from a diseased valve of the left side of the heart, a clot or vegetation, there appears to be little positive proof of a connection between ocular and cardiac affections.

It might be mentioned in passing that there is a possibility of symptoms of asthenopia appearing, coinci-

dent with affections of the heart, which result in a depleted blood supply to the eye. This does not seem improbable, and the suggestion is borne out by the facts presented in subsequent chapters.

KIDNEY DISEASES

Second numerically, but by far the most important in its effect, is kidney disease. As a matter of fact, it is questionable if it is second even numerically. In reality, it is probably by far the greatest cause of death because of being the inciting cause of other diseases, as, for example, in heart disease as mentioned above.

Regardless of the correctness of this surmise, it is unquestionably true that kidney disease and its reflex symptoms enter more largely into optometric practice than any other single disease.

So great is this predominance, especially when taken in conjunction with diseases of the circulatory system, of which apoplexy may be considered a part, and so negligible is the effect, in optometric practice, of the other diseases mentioned, that it will be more profitable to devote the balance of this chapter largely to the former. In justification of this course, it may be pointed out that PNEUMONIA, for example, as a matter of course does not enter into our work. While CANCER may present some of the symptoms of eye-strain, such as digestive disturbances, the probability of a cancerous patient applying to the optometrist for relief from such symptoms appears rather remote. The author has never knowingly encountered such a case, nor have any of his cases been reported later as being afflicted with that disease, altho, of course, there probably have been instances.

It is likewise improbable that the tubercular individual will apply to the optometrist for aid. His symptoms are evidently such that the general practitioner is more likely to be consulted.

This is also true of those suffering from the diseases of the NERVOUS and DIGESTIVE SYSTEMS mentioned in the Life Insurance tables. Included in the list are such disorders as general paresis, epilepsy, locomotor ataxia and other diseases of the spinal cord, paralysis of the insane, hernia, appendicitis, ulcer of the stomach, cirrhosis of the liver, diarrhea, gall stones, etc. The symptoms of these conditions are usually so severe that the need of a physician is at once indicated, so that such cases rarely present themselves to the optometrist.

On the other hand, the milder ailments, such as general nervousness, indigestion, etc., are frequently due to eye-strain and disappear with the application of properly correcting lenses. The border line between the mild and more grave disorders is so ill defined that it is difficult to give an accurate differential diagnosis in each case.

Inasmuch as such a diagnosis is so largely a matter of guesswork by even the best physicians, the optometrist need not feel greatly concerned if he occasionally comes to an erroneous conclusion and prescribes glasses when more heroic treatment is required.

As a rule, the morbid processes resulting from these diseases do not reach the eye, or, if they do, not in sufficient quantities to cause disturbances. Therefore, individuals suffering from them are not likely to feel symptoms which direct attention to the eyes. It follows that in optometric practice it is not so necessary for the practitioner to be able to detect such diseases, as they have little or no direct bearing upon his work, and they

do not frequently present the symptoms he is called upon to relieve. They do not, then, come strictly within the province of preventive optometry. Even ultimately their effect upon the eye is usually negligible.

Of course, the optometrist is not called upon to recognize the diseases mentioned; still less to offer a differential diagnosis. In fact, he should practically never give voice to his opinion except as indicated in subsequent pages.

Notwithstanding this doctrine, there is an opportunity for service with the application of the science of sphygmomanometry in optometry which constitutes a moral obligation for those who care to assume it.

Generally speaking, reference is made to the fact that sphygmomanometric readings sometimes indicate diseases not at all related to the eye, some of those mentioned, for instance. Inasmuch as they may present no symptoms directing attention to the eyes or remediable by optometric measures, it is not improper to disclaim any obligation concerning them. Nevertheless, if, in the course of his examination, the optometrist discovers a symptom causing him to think the patient has such and such a disease, it is conceivable that it would be morally wrong, though perhaps ethically right, not to acquaint the patient with the fact that a physician's examination would be advisable.

Specifically, reference is made to TUBERCULOSIS. All medical authorities seem to agree that this disease has a characteristic low pressure. If such a pressure is encountered in an examination, and the patient shows other symptoms of the disease, would it be charitable to dismiss the patient without a word of warning? The writer thinks not, though he concedes that a logical argument

to the contrary is possible. It is a matter which rests largely with the individual practitioner.

The same reasoning can as well be applied to the other diseases which oftentimes present abnormal pressures. The student's acquisition of knowledge concerning such diseases, by perusal of standard works upon the subject, will indicate the action most advisable for him to take.

Returning to the main subject, it is highly desirable, from all points of view, that the optometrist be able to detect those symptoms of vascular and renal diseases which can be ascertained by the simpler means at his command.

There are three arguments permissible for the support of this statement, viz.: 1. The general physical welfare of the patient. 2. The ocular welfare of the patient. 3. The immediate and ultimate welfare of the practitioner. Let them be considered in that order.

1. GENERAL PHYSICAL WELFARE OF PATIENT. Reference to the tables, and analysis of the final one, indicate that of the total causes of death in 12,439 cases, 35.2 per cent, or 4,378, died from diseases of the kidneys and of the circulatory system. From kidney disease alone, the deaths were 1,281.

It is reasonable to assume that these percentages are applicable, with only slight variations, to the entire death rate of this country, excepting only those occurring among children of five years and under. The enormous total of each year is appalling and leads to the inevitable conclusion that "something should be done about it."

Fortunately, this is entirely practicable, in so far as the matter rests with science, owing to the facts that (a)

the cases of circulatory and renal diseases are largely known; (b) the symptoms of these diseases are generally susceptible of ascertainment; (c) the cure can often be accomplished by following the instructions laid down by the medical profession.

It is neither the purpose nor the province of this work to instruct the reader, comprehensively, regarding these three facts, but a passing survey of them is essential for a proper appreciation of the principles involved in this chapter.

(a) Considering, then, the causes of the circulatory and renal diseases, the author understands the predominant one to be the ingestion of poisonous or otherwise improper food materials. The proposition of undertaking a selective diet is one in which the average individual takes little interest until derangements of the system make it compulsory—and then, as previously pointed out, the damage will have already been done, at least in part. This is clearly shown in Mr. Hunter's remarks concerning the effect of alcohol. His figures constitute conclusive evidence in this respect and demonstrate "beyond peradventure of doubt," that, for instance, it does make a great difference whether the average individual indulges in one, or two or more "drinks" a day.

The writer recognizes no reason why the same argument should not apply to any other voluntary act of the individual involving health and permitting of selection. It is commonly conceded that most individuals eat, sleep, drink, work or exercise insufficiently, or to excess, or otherwise improperly. Since a chain is no stronger than its weakest link, it follows that the violation of any of the rules of health exacts a penalty; if such violation is

persistent, the ultimate result will be in evidence in statistical reports.

But, as elsewhere stated, the average individual will continue to act contrarily to the laws of nature, and will therefore continue to be punished accordingly. He finds it easy to fall into certain deleterious habits which, seemingly innocuous at first, become second nature to him and established, more or less, as an essential part of his existence. The gravity of the ultimate result is not apparent until considerable damage has been done.

The form of this punishment assumes its final aspect so insidiously that, as a rule, it is not evident to the individual until there is a breaking down of some one organ or function. It is, therefore, but a natural sequence that it is merely a question of time before such an one finds himself—or herself—in the need of therapeutical aid, and visits either the general practicing physician or one who specializes in diseases or functional derangements of the organ which happens to be affected.

Aside from improper eating and drinking, and lack of proper exercise, a great factor in the diseases mentioned is syphilis. It would be futile to discuss this disease under this heading, as it is obviously one requiring no comment beyond the statement of fact concerning its prevalence.

The same may be said of other contributory causes of circulatory and renal derangements. It suffices to say that most of them are known.

(b) Concerning the symptoms of these diseases, those affecting the ocular economy are fully treated in subsequent chapters. Other than these, the chief symptom with which optometric practitioners may be concerned consists of the blood-pressure reading. As stated else-

where, so generally is there a sphygmomanometric reading characteristic of these diseases that, in regard to some of them, a certain condition of blood-pressure is almost pathognomonic of the disease.

It is precisely here that the optometrist's opportunity for service enters. He can ascertain, with little effort and with little loss of time, the condition of the arterial system of the patient and, therefore, be in a position to pass upon the presence or absence of the great symptom of the diseases in question.

The opportunity is a unique one. It has been stated that the violator of the laws of nature will eventually find himself in the need of therapeutical aid because of such violation. It may be said that when the effect of such violation reaches the stage where the victim is aware that something is wrong, the condition has progressed pretty far. How much better it would be if it had been detected *before* the aggravated stage was reached!

This is possible with the optometrist. Even though the eyes are not directly affected, it is a most frequent occurrence that the individual suffering from kidney disease will appeal to the optometrist for relief from eye-strain long before he is aware that he has kidney trouble. How satisfactory it is to discover the condition in time to save a patient much sickness in the future, and perhaps add many years to his life! In the final chapter of this work, the frequency of this occurrence is indicated.

Symptoms other than those due to blood-pressure are known, of course, to the physician, who is the one qualified to render a differential diagnosis.

(c) The burden of curing rests entirely with the medical profession. Its possibility when the progress of the dis-

ease is not too far advanced is too well known to permit of argument.

These three correlated facts clearly present an opportunity for service that is unmistakable. A high percentage of the cases which now appeal to the optometrist for relief from ocular symptoms, caused by eye-strain in one of its several forms, are in a condition to receive such service. Nevertheless, regardless of its magnitude, such service, *per se*, is but incidental to the optometrist's work. It is in reality a byproduct of his endeavors to conserve the future vision of the patient. For, as will be seen, these diseases seriously affect the ocular economy in one form or another when in an advanced stage.

Their recognition constitutes a legitimate duty of the optometrist for this reason. People are putting more and more trust in him not only to relieve immediate symptoms of eye-strain in one or several of its many forms, but to guard their ultimate ocular welfare. Therefore, the general welfare of the patient merges gradually with his ocular welfare.

2. *OCULAR WELFARE OF PATIENT.* The chapters on Ocular Pathology present unmistakable evidence of the relation between ocular diseases and diseases of other organs. In this connection the prominence of the position occupied by kidney disease is manifest. The preponderance of kidney diseases, combined with circulatory diseases, as causative factors in intraocular diseases is indubitable.

The enormous preponderance of these diseases, when taken together, over any other single cause of death mentioned in the table (Fig. 1), would naturally lead to this conclusion, but, even so, their frequency in ocular pathology is altogether out of proportion to their numeri-

cal position in relation to the entire number of deaths. For example, while they undoubtedly cause more intra-ocular disease than all other factors put together, they cause only a fraction of the total number of deaths given in the above report—about thirty-five per cent.

The answer is not necessarily that the ocular economy is peculiarly susceptible to vascular-renal disease,¹ altho the delicacy of its tissues gives weight to such reasoning, but rather that the circumstance is due to the peculiar nature of a combination which carries to all parts of the body the toxins resulting from unbalanced metabolism and elimination.

The products of kidney and vascular diseases enter the eye itself and penetrate every part of the ocular economy reached by the blood.

As pointed out in the preceding pages, this is not so true of other diseases, especially of the diseases mentioned in the table. The only one which might be excepted is nervous disease, but here again is encountered the question of whether or not kidney and circulatory diseases are the etiological factors in most nervous diseases. As syphilis may be considered a blood disease, the answer is in the affirmative, and nervous diseases may be excluded, to great extent, as primary factors in ocular disturbances, since they would largely come within the province of kidney and circulatory diseases.

Of the remainder of the diseases included in the table—pneumonia, cancer, tuberculosis and those of the digestive system—they exercise so little effect upon the ocular economy as to merit exclusion from the present consideration as potent factors.

1. Some writers do affirm such susceptibility.

It might be supposed that, of them, gastric disturbances merit some consideration. This would be true under a general consideration of the causative factors of ocular disturbances, but not in the present connection, for it is difficult to see how the morbid processes of the stomach could reach the eye. While it is true gastric disturbances are often associated with ocular abnormalities, it should be pointed out that they are the common *result* of the former and not the *cause*. Besides this, they rarely affect the eye pathologically, tho temporary disturbances may have a passing effect upon the visual functions by affecting the nuclear or central regions of the ocular nerves. Therefore, gastric disturbances, especially in the milder forms, may more properly be considered byproducts of ocular anomalies, and not inciting causes of them.

With the acknowledgment of this doctrine, the predominance of kidney or circulatory diseases as mentioned is unequivocal. Therefore, the unique position of these diseases, their insidious action and their subsequent and deleterious effect upon the ocular economy render it highly important that, when possible, they be detected in their incipency. When this is accomplished, and the proper medical treatment instituted, the diseases will be checked at their source and the patient spared future suffering.

There is no one better situated for this purpose than the optometrist. When patients apply to the physician for aid, they only too frequently have reached a stage from which recovery is only partial or, perhaps, even impossible. On the other hand, it is a common occurrence for them to apply to the optometrist for relief from a comparatively minor ailment when just in the incipient stage of a grave disease.

As will be seen, some of these diseases seriously affect the eyes, sometimes causing blindness, as well as many other disorders. It is expected that this fact alone will point out to the practitioner his duty, but should it fail to do so, or should he voluntarily fail to fulfill his moral obligation to the patient, it may be pointed out to him that the obligation to himself, imposed by the law of self-protection, should encourage such fulfillment. It is pertinent to consider this phase of the subject.

3. *THE IMMEDIATE AND ULTIMATE WELFARE OF THE PRACTITIONER.* Assuming a case in which certain common symptoms of eye-strain are complained of and a refractive and muscular defect of sufficient magnitude to cause them is discovered, if glasses are given and the patient returns in due time and reports that they have given no relief, and further examinations or changes in lenses meet with the same lack of success, what is the natural sequence of events? Obviously, the patient becomes disgusted and goes elsewhere for service.

This is not an uncommon occurrence. It happens with far greater frequency than is realized by the practitioner, who is but superficially acquainted with the ocular symptoms of general diseases and how closely they resemble the symptoms of ocular strain.

There is nothing more natural than for the practitioner to whom certain symptoms have been described, and who finds a defect commensurate with their magnitude, to give lenses designed to correct the defect and relieve the strain and thereby eliminate the symptoms. Since the symptoms of eye-strain can be simulated by symptoms of renal disease, can anything be more reasonable and in accordance with the laws of common sense

than that a practitioner should use every reasonable effort to discriminate between the two? It seems not, and when means for such differential diagnosis are so simple in application, reasonable in cost and fruitful in results, it is incomprehensible why anyone who is made familiar with them should fail to utilize them.

The desire for self protection alone should dictate their utilization. No professional man wishes to lose a client or patient through a fault of his own nor *through that which may seem to be his own fault*. The argument that the optometrist is not supposed to recognize the difference between the two sets of symptoms may be a legitimate one, but, in view of the facts, it is not exactly a logical one. For as soon as an optometrist professes to relieve the slightest symptoms of eye-strain, he therefore makes it obligatory, if for no other reason than self protection, that he be able to determine whether or not the symptoms complained of are caused *by* eye-strain. To be strictly conservative, he otherwise must limit his work entirely to the correction of whatever ocular defects there may be and place the burden of proof upon the lenses given. In following this procedure, not the slightest encouragement should be offered the patient and not the slightest responsibility assumed by the practitioner—other than that the lenses are correct.

Under such circumstances it would be highly unethical, if not immoral, even as much as to suggest the possibility that the lenses given will relieve the trouble. Strictly speaking, the practitioner should, upon the patient's inquiry, confess in every case that he does not know whether or not the lenses will give relief. He must thereby give each patient to understand that he or she is "taking chances" by having him do the work.

This method of procedure may be very necessary in quite a number of cases, but absolute adherence to it in *all* cases would be, to say the least, most unwise. The man who pursues it to that extreme will command but a meager following. It may satisfy him, but it will seldom satisfy his patients.

A patient wants a positive statement when it can reasonably be advanced. He feels that if the practitioner has not sufficient confidence in himself and in the effect of his lenses to be more or less positive, he—the patient—certainly cannot have much confidence in his work.

This confidence can be created if it is non-existent, or strengthened if it is already present, when means are used to differentiate between various symptoms. The operator is encouraged to trust more to his findings, for experience will tell him whether or not he is on the right track.

His positiveness and the ultimate substantiation of his faith in his work will eventually work to his benefit and to the benefit of those who apply to him for aid. On the other hand, if he uses no such means, he cannot be justified in having the same degree of confidence in his work for the simple reason that his judgment will have been proven wrong on an embarrassing number of occasions. Patients will find their glasses non-productive of results, and their physicians will tell them they were not needed at all—and then they will proceed to give the patient that which the latter regards as conclusive affirmation of the assertion—proof. It is not difficult to realize the position into which such a sequence of events places the optometrist. How much better it would be if he used the sphygmomanometer, discovered

the symptoms of renal disease, and sent the patient to the physician! He would thereby gain the respect of the physician and retain the respect of the patient.

Therefore, it would be stupid to neglect any system, method or instrument, within reason, which offers aid to the optometrist in his effort to discriminate between symptoms caused by eye-strain and those caused by other diseases.

Nothing lends itself to this end so much as does sphygmomanometry. In the first place, the method involves no elaborate technique, but its value is rather in direct proportion to its simplicity. It is cleanly; it could hardly be more simple; and it requires, at the outside, not more than five minutes. Secondly, the information it gives is oftentimes of more value than a chemical analysis. Physicians rely upon it in many instances to a greater extent than they do upon a urinalysis or other methods of diagnosis. Some rank it of greater value to the medical profession than any instrument except the stethoscope and the thermometer.

It is believed that the three arguments advanced carry sufficient weight to conclusively establish the merit of the contention that it is highly desirable for the optometrist to be able to detect those symptoms of vascular and renal diseases which can be ascertained by the simpler means at his command.

Each one by itself must prove convincing to a certain type of practitioner. Together, they cannot fail to reach all.

Of course, it must not be understood that the arguments advanced are limited to only renal and vascular diseases. These diseases constitute merely the bulk of those with which the optometrist comes into contact.

There are many others he will encounter from time to time, and of them there are occasionally those which have a very positive influence upon the eye.

Succeeding chapters give as comprehensive an exposition of these diseases in their sphygmomanometric relationship as the province of this work will permit, and those upon Ocular Pathology point out their influence upon the ocular economy.

In those chapters there are included a number of diseases which may seem to possess no characteristic sphygmomanometric reading, but they are cited for the reason that sphygmomanometry is a science not yet fully developed, and who knows but that the scrap of information given may lead to research that will definitely establish a relationship between the disease and as-yet-unknown blood-pressure symptoms?

CHAPTER 2.

ARTERIAL ANATOMY AND HISTOLOGY

Anatomy. The arteries consist of a system of ramifying elastic tubes pervading practically all parts of the body except the nails, hair, cartilages, cornea, crystalline lens and, usually, the ocular humors. Diagrammatically, they closely resemble the trunk and branches of a tree which has an infinite number of intercommunicating limbs and twigs.

The arterial system initiates in the Ascending Aorta, which arises from the upper part of the left ventricle of the heart, thereby representing the trunk of the arboreal simile, and it terminates in the arterioles which anastomose with the capillary system, which in turn anastomoses with the venous system, the latter being, in a diagrammatic sense, the analogue of the arterial tree, except that it, of course, serves to convey blood *to* the heart instead of *away* from it.

Of the general anatomy of the system, this work is not intended to treat, since it can be obtained more completely in any good book upon the subject. It is pertinent, however, to trace the connection between the artery which supplies the orbit and its contents, and the aorta.

The latter, as it rises from the left ventricle, ascends about two inches, arches to the left and backward and

then descends to the lower thoracic region. At the highest point of its arch it gives off a number of branches among which are the right and left common carotid arteries. As the two are practically identical in their course and in their relations to other bodies, except that they supply the right and left halves of the neck and head respectively, it suffices to describe them as one.

The Common Carotid Artery, then, ascends through the neck, beside the windpipe, and divides into the external and internal carotid arteries about on a level with the thyroid cartilage.

The external branch curves slightly upward and forward, and behind the lower jaw begins to throw off the following branches: (1) Superior thyroid to the larynx and thyroid body; (2) Lingual to the tongue and sub-lingual gland; (3) Facial to the face, palate, tonsil and sub-

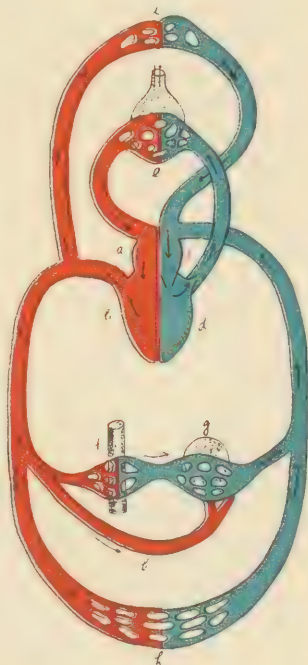


Fig. 2

Scheme of Circulatory System.
(After Tigerstedt)

a, Left auricle; *b*, left ventricle; *c*, right auricle; *d*, right ventricle; *e*, pulmonary circulation; *f*, capillaries of the intestine; *g*, capillaries of the liver; *h*, capillaries of the lower extremities; *k*, hepatic artery. Arterial blood red; venous blood blue.

maxillary gland; (4) Occipital to the sterno-mas-toid muscle and back of the scalp; (5) Posterior auricular to the back of the ear and the adjacent part of the scalp; (6) Superficial temporal to the scalp in front of the

ear and by its transverse facial branch to the back part of the face; (7) Internal maxillary, giving muscular branches to the muscles of mastication, meningeal branches to the dura mater, dental branches to the teeth, and other

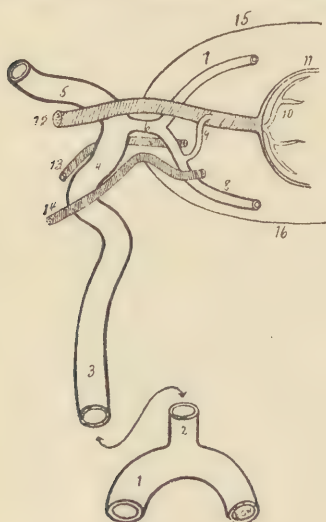


Fig. 3

Scheme of Internal Carotid Artery in its Relationship to the First, Third and Sixth Nerves and the Ophthalmic Artery

1. Aorta. 2. Anastomosis of Carotid Artery with Aorta. 3. Continuation of Carotid Artery. 4. Carotid Canal portion of Carotid Artery. 5. Cerebral portion of Carotid Artery. 6. Ophthalmic Artery. 7. Superior Branch of Ophthalmic Artery. 8. Inferior Branch of Ophthalmic Artery. 9. Central Retinal Artery as it pierces nerve. 10. Central Retinal Artery as it emerges from optic disk. 11. Eyeball. 12. Optic nerve. 13. Third, or oculomotor nerve. 14. Sixth, or abducens nerve. 15. Roof of orbit. 16. Floor of orbit.

branches to the nose, palate and tympanum; (8) Ascending pharyngeal, which gives branches to the pharynx, palate, tonsils and dura mater.

The internal carotid ascends through the carotid canal into the cranial cavity. It "supplies the anterior part of the brain, the eye and its appendages, and sends branches to the forehead and nose. It is remarkable for the number of curvatures that it presents in different parts of its course. It occasionally has one or two flexures near the base of the skull, whilst in its passage through the carotid canal and along the side of the body of the sphenoid bone it describes a double

curve which resembles, somewhat, the letter S placed horizontally. These curvatures probably diminish the velocity of the current of blood by increasing the

extent of surface over which it moves and adding to the impediment produced from friction." (Gray's Anatomy.)

A minute description of this part of the vessel is given by anatomists, but it will serve our purpose here to state that in its course through the carotid canal and the cavernous sinus it is surrounded by filaments of the sympathetic nerves derived from the superior cervical ganglion, thus forming the carotid plexus and cavernous plexus. (Gray.)

Among others, these plexuses give off branches of the sympathetic system to the abducens or sixth nerve and to the ciliary ganglion. The latter transmits these fibres to the radial muscles of the iris, thus influencing the dilatation and contraction of that membrane.

In its course through the cavernous sinus the carotid artery is accompanied by the abducens nerve (Fig. 4), which lies in close relation to it, and the oculomotor and trochlear nerves and the ophthalmic divisions of the trigeminal nerve, which are more widely separated from it. This close relation between the artery and the abducens nerve may well be remembered as it plays a vastly important part in optometric practice, lesions in this area giving rise, in the writer's opinion, to baffling muscle anomalies.

Having passed through the cavernous sinus, the artery rises and passes between the optic and the oculomotor nerves. At this point it gives off the ophthalmic artery which accom-

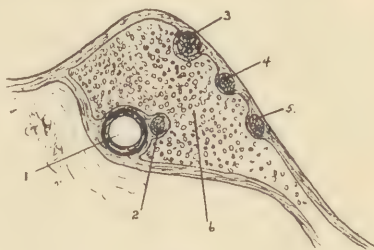


Fig. 4
Relative Position of Contents of Right Cavernous Sinus

1. Internal Carotid Artery. 2. Abducens nerve. 3. Oculomotor nerve. 4. Trochlear nerve. 5. First Division of Trigeminal nerve. 6. Cavernous Sinus.

(Modification of Willson's Drawing in Am. Encyclo. of Ophth., p. 1797.)

panies the optic nerve through the optic foramen and is branched off to supply the various contents of the orbit (Fig. 3).

Of these branches, the central retinal artery is the most important under present considerations. This vessel pierces the optic nerve about 10-15 mm. behind the ball and proceeds forward in the center of the nerve bundle. It enters the ball with the optic nerve and is visible ophthalmoscopically.

This artery is what is commonly called an *end artery*. That is, it does not anastomose or join with other arteries. Usually an artery ramifies with others, thus forming a network so that if one section becomes plugged, it is still possible for the area it supplies to receive blood because its connections with other vessels permit of the section in front of the embolus being filled with blood, and thus the circulation is carried on without serious hindrance.

In the case of an end artery, however, if it becomes plugged, the tissue it supplies atrophies because of lack of nourishment unless the embolus is soon removed or a collateral circulation is brought about by other means. Thus, when the central artery of the retina becomes plugged, the eye becomes instantly blind and remains so if the obstruction is not soon removed. Partial vision may obtain if the so-called cilioretinal arteries exist, but it will be confined to the temporal side of the eye affected. The cilioretinal artery, when it exists, emerges from the optic disk and proceeds toward the macula, being lost in the tissues, apparently, before it reaches that point. It exists in 16-20 per cent of cases, according to well-known authorities.

The retinal vessels are the only ones visible to the eye under normal physiologic conditions, and the variations

in these vessels, when pathologically affected, taken in conjunction with blood-pressure findings, are of the greatest diagnostic importance. This subject is taken up under the section on Ocular Pathology.

Histology.

Arterial histology is of fundamental importance in blood-pressure studies. It is proper, then, to consider the structure of the individual vessel.

An artery is composed of several superimposed coats. The outer coat is known as the *tunica adventitia*. It consists of fibrous, connective tissue. The second coat inward is the *yellow elastic* coat, consisting of elastic tissues and, occasionally, unstriated muscle tissue arranged longitudinally.

The third coat—the so-called *middle*—consists of muscle, elastic tissue and white fibrous tissue. It is sometimes called the elasto-muscular coat. This layer is thin in the smaller arteries, but much thicker in the larger ones. The muscle tissue is in excess of the elastic tissue in the smaller vessels, but this proportion is reversed in the larger ones.

It is in this coat that the vasomotor nerves of the sympathetic system terminate, and it therefore follows that the muscle fibres it contains are involuntary. These fibres are entirely replaced by elastic tissue in the retinal vessels.

Internal to the muscular coat is the *elastic fenestrated* coat, formed of a smooth elastic membrane, perforated by small apertures.

The innermost coat consists of a layer of *endothelial* cells which form the free surface over which the blood

flows. It is continuous with the capillary system, the other coats being discontinued as the artery is transformed into a capillary.

The nourishment of an artery is derived, not from the blood which flows through it, but from small vessels called vasa vasorum, which penetrate its various coats.

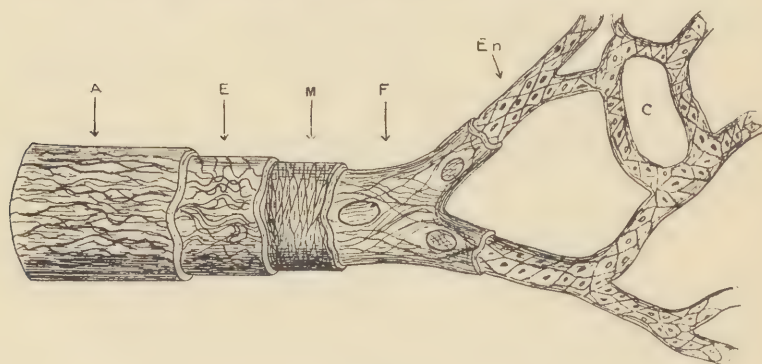


Fig. 5

Diagram of the Structure of an Artery

A, Tunica adventitia; E, elastic coat; M, muscular coat, F, fenestrated coat; En, endothelium continuous with the endothelial wall of C, the capillaries.

These vessels may arise from the artery itself, however, or they may take their origin from an adjacent vessel.

The capillaries are small tubes connecting the arterial system with the venous system. They are from 1-3000 to 1-2000 of an inch in diameter and from 1-30 to 1-20 of an inch in length. As previously mentioned, they do not possess the muscular and elastic coats common to arteries, as these coats are discontinued in the transformation. They join one another and form a vast network over the entire anatomy, with the exception of the parts already enumerated.

CHAPTER 3.

ARTERIAL PHYSIOLOGY

The arterial tree has been described as a system of elastic tubes. The elasticity of the system is perfect inasmuch as the more or less constant change in tension to which it is subjected always results—unless, perhaps, pathological changes take place—in a return to the original volume.

The degree of internal tension to which these tubes can be subjected is enormous when in a healthy condition. Tigerstedt¹ points out that to rupture the carotid of man there is required a pressure of seven to eight atmospheres. As the maximum normal blood-pressure has been estimated to be about one quarter atmosphere, it is obvious that a pressure of about thirty times the normal would be required to cause rupture. Since the normal pressure is approximately 130 mm. Hg., it follows that this internal pressure would amount to 3900 mm. Hg., a figure which clearly denotes how impossible it would be for increased physiological processes to rupture an artery.

It is obvious, then, that the author's suggestion,² that an increased internal pressure brought about by muscle calisthenics might cause an intraocular hemorrhage, has no basis in fact in so far as this reasoning is applied to healthy tissue. It still stands, nevertheless, as a reason-

1. Tigerstedt's Text Book of Physiology, page 202.

2. Keystone Magazine of Optometry, issue of April 23, 1914.

able hypothesis when applied to tissues pathologically weakened. That portion of this work which is devoted to the pathology of the arterial system sustains the contention.

We have seen, from the descriptive anatomy previously elucidated, that the essential elements of the arterial walls are elastic tissue and muscular tissue. The former may well be characterized as a passive agent and the latter as an active one, and they will be considered in the order mentioned.

Assuming an elastic tube, tapering from a wide to a narrow opening, to be filled with liquid and connected by its large opening to a reservoir filled with the same kind of liquid, it follows that if the smaller opening be elevated to a point level with the surface of the liquid in the reservoir, the liquid contained in each will be inert, and exerting no more pressure against the walls than is required by the law of hydrostatics. Let the smaller opening in the tube now be closed and let there be forced into the tube from the reservoir—and the connection between the two closed—an amount of liquid to be designated as one unit, equal to the amount that would flow through the small opening in one second if the *head of pressure* was equal to the aforementioned unit. It follows that since the liquid has no means of escape and is incompressible, it must be accommodated for by an expansion of the walls of the tube. We now have a distended tube possessing in its walls a potential force equal to the amount necessary to force the excessive amount of liquid through the small opening in one second. Now let the small end of the tube be opened and at the same instant let there be forced again into the tube a second unit of liquid from the reservoir, this action being repeated again and again at intervals of a second each.

The action which takes place is this: Upon releasing the small end of the tube, the liquid immediately begins to flow out, being driven by the force potential in the walls of the tube. At the same instant—before an appreciable amount of liquid has escaped—a second unit of liquid has been forced into the tube, thus increasing the tension of the walls. This tension decreases gradually for the period of one second, when again the same thing occurs.

It can readily be seen therefrom that there is maintained a constant flow of liquid through the small opening in the tube, which corresponds to the capillary system of the body, and a constant internal pressure upon the walls of the tube. The tube is never empty and never without a certain internal tension. The liquid it contains is constantly in motion.

This is precisely the action that takes place in the arterial system. The pressure which is constantly present in the walls of the arteries, due to the pressure of the blood column, is termed the *diastolic pressure*, and the pressure which immediately results upon a new influx of blood from the heart is termed the *systolic pressure*. The difference between the two is the *pulse pressure*.

These pressures would be maintained in a constant relation to one another if the passive elasticity of the walls of the artery alone was concerned, and, of course, if pathologic changes did not enter.

However, the system is complicated by the active agent mentioned—the muscular system—and that will now be considered.

It has been mentioned in the preceding pages that the vasomotor fibers of the sympathetic system terminate in the muscular walls of the arteries. These fibers

are of two varieties—constrictor and dilator. Excitation of the one causes a contraction of the walls of an artery, while excitation of the other causes a relaxation.

Proof, susceptible of demonstration, has been offered that between the two sets of fibers there is maintained a constant state of tension throughout the arterial system. This tension varies, however, within somewhat wide limits and according to the laws of supply and demand to which the arteries are amenable. It varies in individual sections and in the relations of different sections to one another.

For example, exercise, resulting in increased katabolism, requires that the volume of blood to the area affected be greatly increased. The action which takes place is a dilatation of the vessels supplying the muscles and skin and a contraction of vessels in other areas. In this manner, it is possible more nearly to maintain a state of equilibrium and to sustain, throughout the entire system, the head of pressure so necessary that all organs may receive sufficient blood to enable them properly to perform their functions.

That this may be properly appreciated let there be assumed a condition in which the lower vessels of the body are dilated to such an extent that they can contain nearly all the blood of the system. Unless a compensatory contraction of the vessels in the middle area takes place and the so-called head of pressure maintained, it is clear that the cerebral portion of the anatomy would have its blood supply so depleted that death would result. As a matter of fact, precisely this condition does take place, it is understood, if a snake is suspended by its head, this creature's anatomy not being so adjusted that a head of pressure can be maintained under all conditions,

as is possible in man. The same condition applies to eels, and also to tame rabbits with large abdomens, if the animal is suspended and immobilized in an erect position from fifteen to thirty minutes. The circulation through the brain ceases, and the heart soon becomes emptied of blood.

The need of such a pressure in man is clearly evident. First, it is necessary that certain force be exerted that the laws of gravity may be overcome and the column of blood raised to the highest point of the anatomy. Second, additional pressure must be applied that, having been raised thus far, the blood is enabled to overcome the resistance of the capillary system and enter the venous system. Otherwise, stasis would result.

Of course this is but a simple explanation and the factors elucidated only minor ones, but they are sufficient to make clear the argument.

The factors which influence the change in pressure, both in the system as a whole and in individual parts of it, are many, and it is needless to elaborate upon them at length. In a person of normal health, the primary factors are (*vide* Tigerstedt): the energy of the heart, the resistance of the arteries, and the total volume of the blood. The secondary factors are, in part, age, sex, physique, exercise, time of day, position, state of mind, amount of liquids or solids ingested, heat, cold, etc. These factors will all be given due consideration in the chapter on Sphygmomanometry.

The widest variations from normal are due, of course, to pathological changes in the vascular system. These will be considered in the next chapter.

Before taking up the study of pathology of the arterial system, it will be well to devote a little attention to

THE PULSE

In the scheme of the elastic tube, mentioned in the preceding pages, the final condition given was that of a tube, distended by a given amount of liquid, with a certain amount flowing out of one end while at more or less regular intervals an equal amount was being pumped in at the other end.

As stated there, this system has its analogue in the vascular system of the human body, and the mechanical changes resulting from the hydrodynamics involved are susceptible of demonstration and yield information of great value.

At the moment of systole, when the left ventricle of the heart contracts, the quantity of blood it contains is thrust upward into the aorta. There it encounters the column of blood contained in the arterial system, which, in comparison with the velocity of the new influx, may be regarded as an inert mass, though, of course, nearly all of it is in a certain state of motion. Since it is manifestly impossible to reduce the volume of this column by compression, as would be the case if it were gaseous, and equally impossible for the heart to force it to overcome its inertia in the brief space of time allowed, it is necessary that some other provision be made. This takes place by a great expansion of the walls of the arteries closely connected with the heart, as illustrated by Fig. 6.

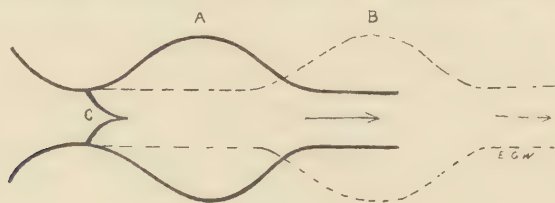


Fig. 6

A. Diagrammatically illustrating the expansion of the arterial walls in response to the influx of blood from the heart through the valves at C, which are now closed. B. The wave progressing throughout the arterial system.

The quantity of blood impelled forth is here piled up, as it were, and exerts a pressure in all directions as soon as the ventricle of the heart begins to expand, the valves in the aorta being closed in the process and held together by the backward pressure of the blood column.

The distended portion of the arterial walls, now exerting a more powerful pressure upon the quantity of blood contained than the section immediately in front of it, begins to propel the blood forward into the next section. (The velocity with which the blood is ejected from the heart is also a factor, of course.) The second section acts precisely as the first, and the result is a wave of pressure throughout the entire arterial tree, altho the extent of the wave is modified to such a degree as the capillary system is reached that the rhythmic change is barely perceptible.

It is this wave that is felt when a palpating finger is pressed upon the radial artery in the wrist. It possesses several characteristics, chiefly among them being (Tigstedt):

1. **Frequency.** The rapidity of the heart-beat varies according to the requirements of metabolism, and is frequently of importance in diagnosis.
2. **Size.** The amount of distention induced by the wave is a noteworthy fact.
3. **Velocity.** The wave can be of either short or long duration. In the one case, there is a sharp, short elevation, and in the other a long, sluggish one.
4. **Hardness.** In some cases it is difficult to obliterate the pulse—comparatively speaking—by pressure, while in others only a slight palpation of the finger is necessary.

These four features will be considered as follows:

1. Frequency. This characteristic depends, as is obvious, upon the frequency of the heart-beat, which in turn depends upon many other factors, such as heat, cold, ingestion of food, exercise, age, sex, psychic influences, etc. Pathologic conditions exert a great influence, of course. The normal rate ranges around 72–75 beats per minute, although Tigerstedt¹ states that pulse-rates as slow as 20–25 beats per minute to as high as 120 per minute have been observed in men of perfectly sound health. It would be safe in such cases to assume the presence of a pathologic condition, accepting the case as healthy only after a process of elimination has been completed.

2. Size. The size of the pulse-beat also depends upon many factors, chief among which may be cited the amount of blood propelled from the heart, and the rigidity of the arterial wall. A large injection of blood into an elastic artery produces a greater distention of the arterial wall than a small one. On the other hand, a rigid artery tends to suppress all pulse-waves and thereby limit the size of the pulse.

3. Velocity. The speed of the pulse-wave is said to average from seven to ten meters per second. This, too, can be said to depend upon the elasticity of the arterial walls. A very elastic wall will expand gradually in response to the pressure exerted, and contract as gradually. In such a case the pulse would have a longer duration than if the walls were inelastic and refused to expand greatly and persisted in contracting quickly. The latter condition would result in a quick pulse-wave. The velocity also depends upon the action of the heart—the

rapidity with which it expels a given quantity of blood. A slowly contracting heart would expel the blood slowly, the result being a sluggish pulse. The converse would be true with a heart which contracts quickly.

Probably nothing so influences the various sound-phenomena encountered in the auscultatory method of taking blood-pressure as does the velocity of the pulse-wave. Since it has been rather definitely determined that it is the changes in the arterial wall which produce the sounds in auscultation, and since the velocity of the pulse-wave exerts the greatest influence upon the rapidity of these changes, the justification for this suggestion can readily be understood.

4. Hardness. This characteristic depends largely upon the internal pressure of the artery. When a high arterial tension exists, it becomes increasingly difficult to obliterate the pulse, as can be readily understood. The natural rigidity of the arterial wall constitutes a factor also, but one not of as great consequence as might be supposed. The resistance of an empty artery, even when sclerosed, is relatively slight to a palpating finger.

Cardiac strength and the volume of the blood expelled have much to do with it, of course. The wave set up by a strong heart is naturally more difficult to obliterate than one set up by a weak heart. A large wave also is harder to suppress than a small one.

Correct interpretation of the radial pulse, however, is not necessarily of optometric interest. In the first place, it is too uncertain, and, secondly, far more accurate and valuable information is obtained by watching the hand on the dial of the sphygmomanometer.

CHAPTER 4.

ARTERIAL PATHOLOGY

Arterial pathology is of threefold importance in the present work. In the first place, idiopathic arterial disease (if the term is justifiable at all) constitutes in itself a serious malady, symptoms of which it is highly important to discover, just as renal disease, heart disease, etc., lend themselves to similar reasoning.

Secondly, the *effect* of arterial disease, idiopathic and systemic, upon the ocular economy deserves a dominating position in any work relating, however remotely, to the eye.

Thirdly, and most important of all under present considerations, arterial abnormalities, whether caused by local or remote lesions, constitute the fundamental basis of sphygmomanometry.

As differential diagnosis of the diseases of any organs or systems other than the eye is beyond the province of this work, the *first* factor may be dispensed with at once. It is not necessarily important that the optometric practitioner be enabled to analyze his discoveries, as that can better be accomplished by those more fitly trained for such work. While it is, of course, important that arterial disease be discovered, if the optometrist succeeds merely in recognizing a *symptom* of such an anomaly, and sends the patient to the medical practitioner, he will be fulfilling

a function far higher than would have been conceded to him not long since. Therefore, in the pages that follow it should be understood that diseases of the arterial system and of other organs of the body (with the possible exception of the eye) are spoken of only in a general way, their relation to the blood-pressure discovered being given more to impress the practitioner with the fact that the blood-pressure test covers a great range of diseases than to instruct him in specific diagnosis.

In other words, if he discovers an abnormal blood-pressure, it is of secondary importance whether the condition is caused by vascular, renal, cardiac, cerebral or neural disease. The primary fact that *something* is wrong has been ascertained, and the determination of *what* is wrong is outside the province of optometric work.

The *second* factor is discussed elsewhere and need not be repeated here.

Notwithstanding the above statement relative to arterial disease, the *third* factor necessitates a concession in the form of a general explanation of the changes in structure which take place when an artery is attacked by disease. The vascular system is in the peculiar position of not only being susceptible to disease itself, but it is also a "common carrier" of the waste products of disease and unbalanced metabolism in other organs. In addition to being a carrier of such products, it is attacked by them. Its variations serve, therefore, not only as an index of its own condition, but of the conditions of several other organs, and its importance is altogether disproportionate to its individual and purely local disturbances. A careful consideration of its variations is therefore justifiable and appropriate.

The most common lesion that takes place would seem to be that which is covered by the, so to speak, "blanket"

term, arteriosclerosis. As may be assumed from the term, the condition presents itself as a hardening of tissue.

The process which leads to this end is, briefly, a degeneration of the media or middle muscular coat of the artery, together with a thickening of the intima or innermost coat. The latter is more or less a compensatory process brought about to maintain the resisting strength of the walls which the former degenerative processes had weakened. It probably also serves the purpose of retaining the original volume of the vessel, since the process of degeneration naturally tends to increase the capacity by distending the walls and thinning the middle coat.

Aside from atrophy and degeneration of the muscle fibers of the middle coat, there also takes place a destruction of the elastic elements. The thickening of the internal coat is said to be due to the development of connective tissue between the endothelium, or inner, coat and the adjoining elastic tissue.

It is patent that these processes lead to the destruction of the elasticity of the vessel. Since this elasticity is the essential characteristic of the arterial system, it can readily be appreciated that its loss can lead to serious consequences. This will become more apparent in subsequent chapters.

There still seem to exist differences of opinion concerning the inciting causes of such changes.

Traube's theory was that it was caused by continued high blood-pressure, and Klotz has sustained the contention by suspending a rabbit head downward and demonstrating a sclerosis of certain vessels which had been subjected to increased tension because of the posture.

Thoma held that the chief factor was a lessened resistance of the media, in consequence of which the vessel became widened and the blood stream slowed, necessitating the growth and thickening of the endothelial lining that the relation between the artery and its contents might be maintained. It is evident that the two theories can be interdependent.

Both conclusions seem logical, and this is evidently the opinion of Sir Clifford Albutt who divides such cases into three classes: 1. Hypertension cases. 2. Cases which have a toxic origin. 3. Cases which may be termed idiopathic; therefore, truly sclerotic. In the first, the tension *causes* the disease; in the second, an infection causes the symptoms, which may therefore be transitory; and in the third, the high pressure is the *result* of the disease.

Consideration of the primary class naturally gives rise to the thought: What causes the high blood-pressure in the first place if the artery is in a healthy condition? The reasons for such a condition necessitate so deep a study of pathology in general that it would be out of place to fully introduce them here. Furthermore, the author does not claim to be competent to discuss them comprehensively.

Nevertheless, the condition is so obviously one brought about by the excitation of the eliminative processes that this can be stated, without hesitation, as the fundamental cause.

Liquid which is injected into a system of tubes in a manner similar to the injection of blood into the arteries, flows through them more rapidly if the tubes are reduced in caliber and rigid instead of elastic, the cause being that the force of the pump is directed more nearly against the

entire column of liquid instead of against only that section immediately adjacent to the valves of the pump. Further acceleration occurs automatically if the liquid is pumped in more rapidly.

When the tearing-down processes of the tissue occur more rapidly than usual, thereby liberating an undue quantity of waste products, or improper functioning throws into the blood stream an amount of unassimilated material larger than is consistent with health, it becomes the duty of the blood to eliminate these poisonous products as rapidly as possible.

This it can do by carrying them with increased speed to the organs which aid in such elimination. The acceleration is accomplished by stimulus of the vasoconstrictor muscles which cause the vessels to assume more nearly the properties of inelastic tubes. It is further aided, no doubt, by accelerated action of the heart. Naturally, this rigidity results in greater resistance of the walls to the blood column, and a higher internal pressure is the consequence.

It is very likely, too, that increased rapidity of flow enables the blood to retain, until the proper organ is reached, those organisms or materials which would otherwise find lodgment in the interstices of the capillary walls and bring about degenerative changes in adjoining tissues. The author is not aware that this hypothesis has been advanced in medical literature, but it is so simple and logical that it probably has.

It is perfectly natural that if the hypertension is maintained for an abnormal length of time, pathological processes take place, since it is apparently the rule that sustained muscular action results in hypertrophy of the muscle involved. This is probably Nature's provision

to relieve the nervous exhaustion that would otherwise result. Of course, the high tension which terminates in sclerosed arteries may have its origin in causes other than the one elucidated, but this suffices to acquaint the student with the mechanics involved.

The second class, that which includes those cases due to toxic origin, needs little explanation other than that already given. It is a matter of course that toxic or infectious substances tend to degenerate any organs they attack.

It can be mentioned, however, that of the diseases that lead to degenerations of the vessels, syphilis is perhaps the most important. (R. H. Babcock: Diseases of Heart and Arterial System.)

Indeed, it might serve a good purpose to digress for a moment and point out that this disease seems to exert a detrimental influence in an enormous number of organs and ailments. Medical literature is thoroughly permeated with references to it, and practitioners in all therapeutical sciences would do well to bear in mind the prevalence and influence of the disease. It is perhaps particularly important to the optometrist because of its effect upon the ocular muscles. Indeed, one writer (F. W. Mott) goes so far as to say that paralysis of the third nerve can be regarded as almost pathognomonic of syphilitic disease. The same author says further, "Diseases of the arteries of the central nervous system, occurring in a person under forty, is generally due to syphilis, the virus of which produces an inflammation of the coats of the vessel, especially the inner."

Chronic alcohol, lead and tobacco poisoning may also be mentioned as etiological factors.

The third class of cases in which the high pressure is the *result* of the disease may be considered partially as a sequence of cases in the second class and more or less dependent upon them. Nevertheless, it should be pointed out (*vide* Babcock) that this vascular change may be brought about by reason of a decrease in the volume of the blood, caused by anemia, hemorrhages, etc. The vessels, in a case of this kind, would naturally attempt to compensate for the decrease in pressure by narrowing their lumen, thereby decreasing their volume and increasing their pressure. This change would be accomplished by thickening their intima.

This is then a spontaneous alteration of the tissue and may be termed "idiopathic." Its progress evidently depends upon the general health of the individual and the care exercised in eating, drinking, etc.

Probably the factor exercising the greatest influence in such cases, a factor which hardly can be called pathologic, is age. There is a gradual rise in pressure with advancing years, owing to senile changes in the arteries. The change is remarkably similar to the change in the amplitude of accommodation of the crystalline lens and probably is due to identical causes. These variations will be more thoroughly discussed in the chapter on Sphygmomanometry.

It is not pertinent to elaborate upon the effect of renal and cardiac diseases upon the arterial system, but it can be mentioned in passing that they apparently exert more influence of a pathologic nature than any other single factor (*vide* chapter on General Considerations). Certain it is that cardiac, renal and vascular abnormalities play a greater part in sphygmomanometric readings than any others, perhaps more than all the rest combined.

This is particularly true of cases displaying hypertension. After observations taken of several hundred high pressure cases, Fischer announced the presence of kidney disease in over sixty per cent of the number of those whose pressure ran over 140 mm. Hg. Eighty per cent gave evidence of chronic kidney changes.

The variations in blood-pressure that can take place under pathological influences are very great. It has been reported (Neu) that patients have recovered after sustaining a pressure as low as 50 mm. Hg. Just how high a pressure can be sustained it is difficult to say. Cases running considerably above 300 mm. Hg. have been reported.

The normal physiological pressure in cases over twenty years ranges from 100 mm. to 150 mm. Hg. These figures have been arrived at by the medical profession, authorities of which contend that any continuous pressure outside these limits is pathological. The writer finds, after some experience, that in optometric work the lowest figure should be 110 mm. instead of 100 mm., thereby giving less elasticity to the normal variation permissible.

The reason for this is that patients having a continuous low pressure are usually people with reduced resisting power. Such cases are often so constituted that a very slight optical defect produces the graver symptoms of morbid functioning, and it is frequently a question whether or not correcting lenses will give relief.

Furthermore, even if relief is given at first, the usual confidence in its permanency is not justifiable, as such constitutions are too vacillating and insecurely founded

to withstand the mild assaults of fatigue, nervous disturbances, etc.

Therefore, a pressure under 110 mm. Hg. should be regarded with suspicion, and a most conservative prognosis ventured. This subject is dealt with more specifically in subsequent chapters.

CHAPTER 5.

SPHYGMOMANOMETRY

In Chapter 3, where the physiology of the arterial system is explained, it is pointed out that two fundamental pressures exist in relation to the arteries and the blood they contain. These are the *systolic* and *diastolic* pressures. The third pressure, the *pulse* pressure, is mathematically determined by the other two, as previously stated.

At the place mentioned it was explained that the systolic pressure, sometimes called the *maximal* pressure, exists at the moment the heart has succeeded in forcing into the arterial system the quantity of blood contained in the left ventricle. It is clear that the addition of this quantity of blood, variously estimated at 50 to 100 cc. (Warfield), to the quantity already present in the arterial system, necessitates its accommodation by an expansion of the walls of the arteries, since there cannot be an instantaneous outflow of an equal amount through the capillary system.

It necessarily follows that since the arteries are highly elastic they have a tendency to contract to their former size, and this tendency results in there being brought to bear upon the blood column an increased pressure which is more or less in direct relation to the elasticity of the walls of the artery and to the amount of blood forced into

them by the heart. This pressure, which is the result of the *increased* pressure combined with the pressure *already existing*, is known as the systolic pressure, as it takes place *immediately following* the moment of systole.

It has already been explained that there always exists a physiological pressure throughout the arterial system in order that the blood may overcome both friction and gravity and thereby be forced through the capillary system. The lowest point of this pressure occurs when the contracting arteries have succeeded in forcing through the capillaries an amount of blood about equal to the amount forced into the aorta by the heart. Naturally, in an elastic system this is a comparatively gradual process and it reaches its greatest extent just *preceding* the new influx of blood caused by the contraction of the heart. The pressure existing at this instant is the diastolic pressure, sometimes known as the *minimal* pressure.

Sphygmomanometry is the science of the measurement of these two pressures. Essentially, it consists of the application to an artery of a pressure sufficient to close the artery and prevent the passage of blood through it. This pressure is measured by noting its power to raise a column of mercury in a glass tube a given number of millimeters, or by an equivalent of this process.

The artery commonly employed for this purpose is the brachial, situated in the upper arm between the elbow and the shoulder and on the inner side toward the body. In its course at this point it runs beside the humerus bone and therefore has a solid body against which it can rest as pressure is exerted.

Several methods have been devised for the purpose of achieving this end, such as employing simple weight

pressure (at a point other than the one mentioned), water pressure and air pressure, but the last mentioned has been found the most practical in all respects. The appliance necessary consists of a flat, oblong, rubber bag, 12 cm. in width, enclosed in a non-stretchable cloth covering which is continued for two or three feet as a bandage.

Projecting from the bag are two rubber tubes, one of which is connected with a rubber bulb inflator, and



Fig. 7

Showing the Bandage Which is Applied to the Arm

the other with the manometer, whether the latter be of the dial or mercury type.

The part of the sleeve containing the bag is placed snugly on the inner side of the bare arm, and the remainder of the cloth wrapped around and around in the form of a bandage, the last few inches being tucked under the preceding fold.

The air is now pumped in by means of the syringe bulb, and the bag slowly inflated. As the pressure gradu-

ally rises, the brachial artery is gradually obliterated and the blood stream shut off. The pressure is communicated to the attached instrument and registered in the form of millimeters.

TECHNIQUE

Before explaining how the operator determines the maximal and minimal pressures, it is necessary to point out that a certain technique is necessary to the above procedure in order that success may be insured.

It is important that the bag be placed upon the *inner* surface of the arm and *on a level with the heart*. If it is placed higher than the heart, the sphygmomanometric reading will be lower than it should be, and if it is placed lower, the reading will be too high, owing to the difference in the sheer weight of the column of blood in the artery above the armlet.

In regard to the necessity of the arm being bare, it is, of course, advisable that this be the case, but the writer does not believe it absolutely necessary in all cases in optometric practice. In the first place, he has found very little variation in the manometric readings when the bag is applied to the bare arm and when applied to an arm covered with either the shirt sleeve or the sleeve of an under garment, or both. Secondly, it is sometimes impossible to uncover the arm to the shoulder without placing the patient under great inconvenience and embarrassment, and the factors so introduced, psychic and otherwise, tend to defeat the purpose of the examination. Furthermore, the slight variations which may be found under the two conditions are usually unimportant in optometric work, tho they may well be of vital interest to the physician. The major variations from nor-

mal are the ones the optometrist more greatly desires to ascertain. However, as will be seen, it is essential in most cases to have the arm bared to a point 2 or 3 cm. above the elbow, or sufficiently for the application of the stethoscope.

Having applied the bag and bandage as suggested, the arm (usually the left arm) is extended outward from the side at an angle of thirty to sixty degrees and rested upon a table, with the forearm horizontal. All muscles are to be relaxed to their fullest extent, the arm lying perfectly free from strain. The patient should be assured in a casual manner that the procedure is a simple one, not requiring more than two or three minutes, and entirely free from pain. Otherwise, if concern exists, it is possible for it to exert a psychic influence and so affect the manometric reading.

The sphygmomanometer and the bulb are then attached and all is in readiness for the blood-pressure determination. There are two principal methods employed for this purpose and they are here explained in the order of their simplicity.

PALPATION METHOD

When this method is used, the patient's wrist below the bag is grasped by the operator, and either one, two or three of his first three fingers applied to the radial pulse, which is usually found without great difficulty. There may be a question as to how many, or which, of the fingers mentioned should be used, and usually authors seem to incline to the first and second. The writer has found that in his individual case the balls of the second and third possess greater tactile sense than that of the first, and therefore more quickly and accurately register a change

in the pulse-wave. It is a matter to be decided by the individual after experiment.

With the fingers now placed over the pulse at its strongest point, the bag is more or less slowly inflated, and the hand on the dial of the sphygmomanometer, or

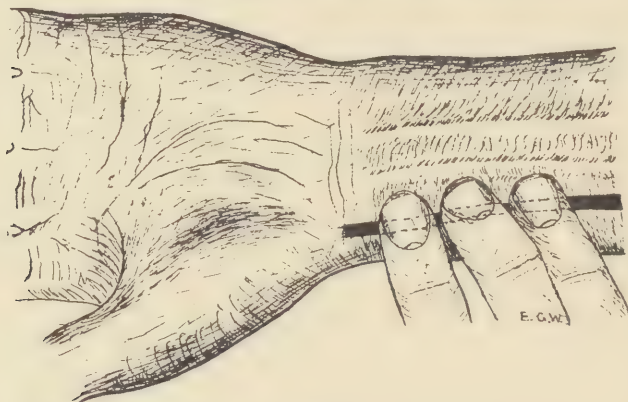


Fig. 8

Diagram Illustrating Application of Fingers to the Radial Artery at the Wrist

the mercury in the glass tube, watched, while close attention is being paid to the pulse at the wrist. *When the pressure in the bag reaches a point where the pulse at the wrist ceases, the reading then given by the manometer is noted and the pressure forced 10–20 mm. Hg. above that point.* The release in the bulb is then pressed upon and the air gradually permitted to escape, reducing the pressure not more than 2–4 mm. Hg. at a time. *When the first pulse-wave is felt at the wrist, the registration of the instrument is noted, and the lowest point of the excursion of the hand constitutes the systolic pressure.*

For example, assume a case in which the pulse at the wrist ceases when the instrument registers 125 mm. The pressure is now forced to about 140 mm. and then slowly released. At about 125 the hand on the dial (assuming

an aneroid instrument is being used) is making (usually) excursions of 2 to 6 mm.—from 125 to 127 or to 131 mm. Hg. If the first pulse-wave is felt at this moment, the systolic pressure is 125 mm. Hg. and *not* 127 or 131 mm.

For determining the systolic pressure this method is a reasonably accurate one, the writer finding it to differ but slightly from the auscultatory method, which will be explained and which is regarded as a criterion. There is usually a difference of 2–5 mm. Hg., but in the average systolic reading this is not sufficient to cause concern to the optometrist.



Fig. 9

Cross-section of wrist, showing how the palpating fingers can be rested upon the edge of the radius bone. If excessive pressure is unconsciously applied, it is directed against the bone instead of against the artery, thereby preventing obliteration of the latter.

Occasionally the difference amounts to 10–20 mm. Hg. and therefore, to be on the safe side, it is preferable to use the auscultation method whenever convenient. Some one has well said that in nine cases out of ten the palpation method is exact, but that the tenth case is the important one.

For determining the diastolic pressure the palpation method is absolutely undependable. It has been suggested that this pressure could be ascertained by noting the point at which the pulse at the wrist became the widest and strongest as the air was released from the bag

after the systolic pressure was taken, but it is manifestly impossible accurately to determine this point by the tactile sense. Even delicate instruments which register this change upon a revolving drum have been found unreliable for the purpose.

The same can be said of the oscillatory method of determining the diastolic pressure, in which the hand upon the dial of the sphygmomanometer is observed. As the pressure is reduced, the hand oscillates in response to the pulse. The reading *just after* the widest excursion of the hand is supposed to represent the diastolic pressure. In the first place, it does not seem to have been conclusively proven that the widest excursion of the arterial walls truly represents the diastolic pressure period, and, secondly, it is quite difficult accurately to determine visually this point. Therefore, the method cannot be recommended.

In using the palpation method, the writer has found it advisable to have his arm and hand *inclined downward from the palpating fingers to the elbow*, as, otherwise, the impact of the blood in his own arteries, as it rushes downward in obedience to both gravity and the heart's contraction, causes a very perceptible pulse in the fingers of his own hand and there is danger of mistaking this for the return of the pulse in the patient's wrist. The first few return waves are very weak and their detection requires the elimination of confusing factors as well as a well-developed tactile sense.

AUSCULTATORY METHOD

The *sound* sense instead of the *tactile* sense is utilized in this method, and the results have been found to be much more reliable. In ascertaining the systolic pressure

the possibility of error, even of 2 or 3 mm. Hg., is reduced to the minimum, and it is the *only* method by means of which the diastolic pressure can be accurately determined. With it, the latter pressure is as easy to find as the former one.

While a thorough understanding of the auscultatory method necessitates intricate reasoning and a full knowledge of the involved phenomena, its application in practice is comparatively simple. The only instrument employed in addition to the sphygmomanometer is the stethoscope.¹ In auscultation, the arm band is applied in the manner previously described, care being taken that 2 or 3 cm. of the bare arm is exposed between the elbow and the lower edge of the bag. The bag is inflated in the usual manner, and the bell of the stethoscope applied to the bare space below the bag as illustrated in Fig. 10.

It will be noted that this space is located just above the elbow, toward the inner side of the arm, between the internal condyle and the internal border of the biceps. The course of the artery to and under this point can usually be traced by palpation, the fingers being pressed firmly upon the forearm, just below the elbow, until the pulse is felt. Upon gradually moving the finger upward, it will be found that the pulse shifts to the inner side of the arm, and finally becomes lost, to ordinary pressure, in the surrounding tissue.

Occasionally, in fat arms particularly, it is difficult to hear sounds in this region, or difficult to place the bell of the stethoscope in the proper position. In such instances, the upper part of the forearm, where the pulse is easily palpable, just below the elbow, serves the pur-

1. The Singer stethoscope is used by the writer and has been found very satisfactory.

pose better, the sound being fairly well heard at this point.

As the pressure in the bag is raised, it will be noted that a more or less clear thump is heard, coincident with

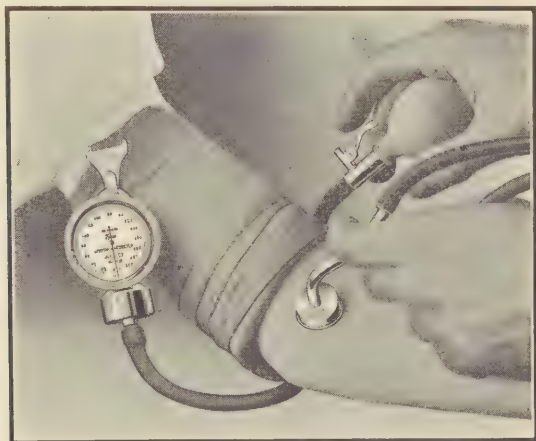


Fig. 10

According to anatomists, the Brachial artery bifurcates into the Radial and Ulnar Arteries about one-half inch below the bend of the elbow. While it is better to apply the stethoscope as indicated, there may be occasions when this is inconvenient, owing to shortness of upper arm, or to the bundle of clothing present when sleeves are rolled up; in such cases the writer has found it convenient to apply the stethoscope bell just below the elbow, at or above the point of bifurcation. Good results can be thus achieved.

the heart-beat, as the pressure reaches 70-90 mm. With more or less change in character, this sound continues until perhaps 120-130 mm. is reached, when it disappears. The pressure is elevated 10-20 mm. above the point of this disappearance, just as in the palpation method, and then slowly released in the manner described under that method.

Assuming the case to be one in which the systolic pressure is 125 mm. and the diastolic 85 mm., as the pressure is lowered toward the former figure it will be

noted that the hand on the dial of the instrument begins to oscillate with the heart-beat. This oscillation is to be carefully watched as the hand slowly revolves backward. As 125 mm. is approached, the movement of the hand executes wider excursions and at just about 125 mm. a clear *thump* is heard with the stethoscope. This sound registers the systolic pressure which is taken at the *lowest point* of the excursion of the hand, just as in the palpation method.

The stethoscope is still held in its position and the pressure in the bag slowly reduced. For perhaps 12 mm. this clear thump is continued, after which, for about 15–20 mm., it becomes transformed into a so-called murmur, which is very easily interpreted as spurts of blood through a somewhat constricted artery.

At about 92 mm. this sound again becomes transformed into a clear, snapping thump which lasts until 85 mm. is reached. At 85 mm. it becomes altered into a dull thump which lacks the resonance of the previous sound and which continues for perhaps 5 mm., after which all sounds disappear.

The point where the *second snapping* sound becomes transformed into the dull thump—*i.e.*, 85 mm.—represents the diastolic pressure.

There are then five so-called phases to auscultation, occurring in the following order:

First phase. The clear snapping sound after the pressure has been reduced from above the systolic pressure, this sound representing the true systolic pressure.

Second phase. The period during which the murmur is heard, caused by the whorls or jets of blood through a flattened artery.

Third phase. The second clear snapping sound, the termination of which represents the true diastolic pressure.

Fourth phase. The dull thumping sound, very similar to the sound given when a punching bag is struck or a football kicked.

Fifth phase. The disappearance of all sounds.

ANALYSIS OF AUSCULTATORY PHENOMENA

(The following, to page 64, may be omitted by those who do not wish to indulge in the deeper study of auscultatory phenomena.)

Careful analysis of these phases and the conditions involved encourages the following conclusions:

1. In the first phase, the semi-clear snapping sound is caused, perhaps, not so much by the sudden opening of the artery under the cuff as by the sudden relaxing of the negative tension under which it has been placed by the compression of the cuff. It must be remembered that when the blood flow in the artery under the cuff is shut off, the artery itself is not merely relaxed; it has been forcibly compressed into a smaller volume than it would naturally assume were it relieved of all internal and external pressure. Because of the fluid contents of the arm, pressure has been exerted from all sides, as indicated by the following diagram:

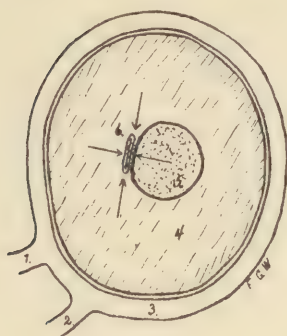


Fig. 11

1. Tube leading to manometer. 2. Tube leading to inflating bulb. 3. Rubber bag surrounding arm. 4. Arm. 5. Humerus bone. 6. Artery upon which pressure is being exerted from all sides.

Therefore, when the pulse-wave is forced through, it is this pressure which must be overcome before the artery becomes actually relaxed, and not the natural resiliency of the vessel. That the crest of the systolic wave succeeds in doing this is evident from the involved phenomena, to wit, radial pulse and first sound phase.

It is patent, then, that for an instant the internal pressure equals or exceeds the external pressure, and the walls of the artery assume a relaxed, neutral position or are even thrown into a position of positive tension. Naturally, this sudden change in an elastic tissue results in sound.

The period during which the internal pressure equals the external is so brief that it is practically incalculable and, therefore, there is no coincident sound of flowing blood as occurs in the second phase.

Some authors argue that the sound is produced solely by the expansion of the vessel due to the sudden influx of blood, but the foregoing hypothesis appears more logical to the writer.¹

1. See Note 2, page 244.

Again, it has been mentioned that the sudden rise in pressure is directed against the bag, and this may suggest the thought that the sound is caused by the resonance of the bag, but McWilliams and Melvin¹ have found that this hypothesis is incorrect. The same authors² have more or less conclusively proven that it is the change in the artery under the armlet which causes the sound, and not the sudden distention of the artery distally to the armlet as is commonly believed and as so many writers assert.

However, it is of little moment which is correct, since all are agreed upon the main proposition—that the first thump is the index of the systolic pressure.

2. The second phase, during which the jets of blood being forced through the artery are heard, is easily understandable. At this point the external pressure has been reduced to such an extent that the vessel is open for a perceptible length of time—sufficiently long for a considerable quantity of blood to be injected into the semi-full artery beyond. Naturally, this small stream, propelled at high velocity through a small opening, produces a churning sound as it penetrates the comparatively quiet pool in the distal artery.

3. In order to simplify the explanation of the diastolic pressure, it is deemed advisable to divide it into two parts, as illustrated in Fig. 12, *c* and *d*.

Fig. 12*c* represents the relative form of the artery when the first thump is heard as the pressure is lowered and the third phase entered into. As stated, the inner circle represents the partially full artery during the diastolic period, when there is a constant flow of sufficient

1. Heart, Vol. V, No. 2, page 184.

2. *Loc. cit.*

blood to prevent the spurting sound of the second phase; the outer circle represents the completely full artery which now has an internal pressure exceeding the external pressure and which state occurs during the height of the systolic wave.

At this time the artery is undergoing three stages: (a) the period of *negative* pressure when the external pressure exceeds the internal pressure; (b) the period of *neutral*

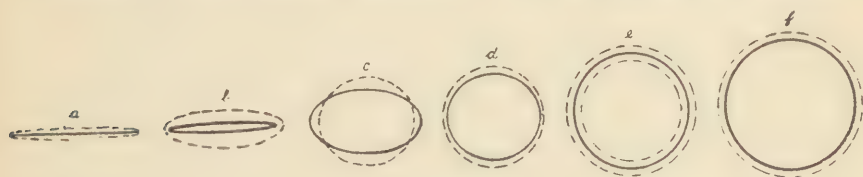


Fig. 12

Illustrating Relative Positions of Arterial Walls During the Several Phases of Auscultation

The solid lines represent the position of the wall during diastole; the broken lines, its position during systole.

a, First phase; *b*, second phase; *c*, early period of third phase; *d*, latter period of third phase; *e*, fourth phase; *f*, fifth phase.

pressure when the internal and external pressures just counterbalance one another and the artery is in a state of rest and free from all stress except that which is the result of the pressure which is applied from all sides equally; and (c) the period of *positive* pressure when the internal pressure exceeds the external pressure and there is potential energy in the walls of the artery which is directed to the center of the vessel.

The first thump occurs at the instant the artery passes from the *neutral* period into the period of *positive* pressure, for it is then that sudden stress exists in the arterial wall and vibration is set up. The reason there is not a second thud as the tension relaxes during the diastolic period is because the walls must first expel the blood the vessel contains, so their relaxation is too

gradual to set up vibration. The gradual decrease is plainly evident by the movement of the hand on the dial of the sphygmomanometer.

Fig. 12*d* represents the final stage of the third phase at the point the diastolic pressure is registered. The inner circle is at a pressure just less than or equal to the external pressure, and the outer circle represents the upper limit of the systolic wave.

While the former is given as depressed a trifle, it is not necessarily so actually; it might either be slightly depressed or it might be perfectly round without altering the value of the argument. The point that must be borne in mind is not that it is the shape of the artery which counts, but rather the introduction of its natural elasticity. The former is incidental.

During this period, all pulse-waves register a clear snapping thump because the elasticity of the arterial walls is brought into play by their sudden expansion—by their sudden change from a state of laxity or neutral pressure into a state of tension or positive pressure.

When there is a further reduction of the external pressure, the snapping thump ceases because there is no longer a change in the character of the arterial wall; the pulse-wave merely augments a state already brought about by the change in relation between the internal and external pressure. The pulse-wave merely expands further a vessel already expanded. Hence, a reduction in the sound.

The change in the arterial sound from a clear thump to a dull one registers the diastolic pressure simply because it indicates that *between the two sounds the internal pressure and the external pressure have been rendered equal.*

4. After the internal pressure during diastole definitely exceeds the external, but only by a slight amount, there still occurs a slight sound which soon disappears as the difference in pressure is increased. This sound corresponds to the fourth phase.

It seems possible that this last sound occurs while the excursion of the arterial wall is still within the range of its normal elasticity and ceases when the artery is dilated to the fullest extent of its natural elasticity during the diastolic period. When the latter extent of dilatation is reached, further expansion results in *strain* of the vessel walls, and before the tissues yield to this strain they will offer great resistance. In yielding, they absorb the shock of the pulse-wave without vibrating and, therefore there is no sound.

The hypothesis is substantiated by the changes which occur in the arterial system as explained under Arterial Pathology, *i.e.*, when abnormal pressure exists and is sustained, sclerosis of the arterial walls soon takes place, and even if such a pressure does not exist, the strain to which the walls are subjected under normal conditions causes, in most instances, a progressive sclerosis as the years pass.

Fig. 12*e* illustrates the relative position of the arterial walls in the fourth phase during systole and diastole. The inner broken circle represents the position the artery would assume if the external and internal pressures were equal; the solid circle represents the actual position of the vessel, and the outer broken circle the outer limit of its natural elasticity.

The inner circle of *f*, Fig. 12, represents the outer limit of the natural elasticity of the artery and the position it assumes when practically no external pressure is

exerted, and the outer circle represents the *strained* position the artery takes during systole. This corresponds to the fifth phase, or the phase of no sound.

This gives, the writer believes, a clear and comprehensive explanation of the various sounds that are heard in the arterial wall during auscultation. Of course, it must be remembered that these sounds are not always as clearly defined as this explanation would indicate, but in the majority of instances they follow the rule very closely. The operator soon learns to make allowances for the differences that are encountered. It is natural that such differences should exist when there are considered the variations in the power of the heart, in the quality of the vessel walls and in the amount of fat and other tissue the sounds are to be heard through.

Regarding the first consideration, while it might be thought that information leading to a correct interpretation of cardiac condition concerns only the physician, the writer is arriving at the conclusion that the state of cardiac energy and health vitally concerns the optometrist in the practice of his profession. Tho not in a position at the present time to advance a well supported hypothesis, there are reasons to believe that the correlation between cardiac weakness, inadequate blood supply, ocular (arterial and venous) stasis and retinal and muscular asthenopia is sufficiently intimate to justify an association on the basis of the law of cause and effect. The writer believes there to be a natural affinity between the first and the last of the connected conditions and, furthermore, that while as a matter of course relief of the former should eliminate the latter, local treatment of the light entering the eye will do much toward allaying the

unpleasant symptoms. This hypothesis will be presented in full when conclusively established. The other pathologic influences of cardiac diseases upon the eye have been cited in the proper place under the Diseases of the Circulatory System.

The sphygmomanometric evidence of cardiac conditions is frequently apparent in the intensity of sound heard with the stethoscope. The first phase will be heard as a very weak, faint sound when the heart is weak, and as a more or less loud, strong thump when the heart is strong.

The second phase may be heard either as a mild thump, only slightly different from the first phase, or it may possess the churning quality so characteristic of this phase.

The third phase is the one offering perhaps the most characteristic evidence of the variations that can be expected. Here the stethoscope may yield anything from no sound at all to a loud, reverberating, snapping thump so strong that one instinctively draws away from it as too pronounced for aural comfort.

It is this phase which Warfield regards particularly as indicative of cardiac conditions, stating that weakness of sound is evidence of a weak heart, while loudness of sound indicates a strong heart. The logic of this conclusion is at once obvious when the cause of the third phase is considered. A strong propulsion of blood is necessary rapidly and widely to expand the arterial wall from a neutral position to one of positive tension; and, of course, the power of the heart is more or less responsible under such circumstances. A weak heart could not produce an expansion sufficiently rapid and wide to cause a loud sound, and so there will occur, in such instances, a

weak, low thump. In exceptional cases even this sound will not appear, the second, third, fourth and fifth phases all occurring as one—the last mentioned. In such cases the blood-pressure may or may not be low.

Usually the change from the third phase to the fourth is abrupt and unmistakable. The loud sound is replaced by a very dull thump possessing no sharpness at all. In cardiac weakness it may almost instantly subside and disappear, or, when the heart is strong and other conditions are favorable, it may last through a twenty or thirty mm. reduction in the pressure. However, there are cases in which the change from the third to the fourth phase is less marked and must be listened for closely. Careful attention will usually disclose the point of change.

A sclerosed artery, being more of the nature of a stiffened tube, naturally will yield more sound than a soft, distensible one. The reverberating, sharply-defined sounds may be looked for in such cases, especially when the patient is elderly and possesses a moderately high blood-pressure and a strong heart.

Of course, layers of fat over an artery tend to dull the sound, and when a thick arm is encountered the operator must be prepared to listen very carefully. All sounds will be lessened, but especially the second, third and fourth. Warfield states that the size of the arm does not, as believed in some quarters, influence the blood-pressure reading except in the manner mentioned. The readings are just as accurate as in thin arms. At first glance it might seem as though thickness would tend to heighten the reading, owing to the mass of tissues to be compressed before the artery is reached, but, on the other hand, it should be borne in mind that a high percentage of this tissue is water or other liquid and, therefore, practically incompressible. When viewed from this point, the

proposition merely resolves itself into a matter of hydrostatics—pressure exerted upon a liquid is transmitted in all directions without loss.

Elsewhere it has been stated that sclerosed arteries tend to become hard and of the so-called "pipe stem" variety, meaning, thereby, an artery which feels, under the finger, as a more or less hard, rigid tube. It may occur to some that an artery of this type may itself, through sheer rigidity, offer resistance to pressure, and that when this resistance, as well as the blood-pressure itself, must be overcome, too high a reading is obtained.

Such is actually the case, as the experiments of MacWilliam and Kesson¹ tend to prove. These observers took the blood-pressure of each arm and sometimes found differences as high as 95 mm. existing between them, these differences disappearing almost, if not quite, entirely after several compressions had been made in rapid succession. The change in relationship seemed always to consist of a reduction of the higher pressure until it about equaled the lower one. Their conclusion was that the differences were caused by differences in the rigidity of the arterial wall. They seemed confined almost entirely to elderly individuals of sixty or more.

In all probability great differences are not frequent, and when they do occur, the higher pressure is usually obtained in the right arm, in which the vessels are more likely to be more sclerosed, owing to the greater amount of physical work having been done with that arm. As the pressure should always be taken on the left arm, error due to this condition can be avoided.

Nevertheless, while it is true that in the average case the brachial artery is, in a manner of speaking, perfectly

1. Heart, Vol. IV, No. 3, page 310.

collapsible, it has been the writer's experience that nearly always a slightly lower pressure is obtained—2 to 10 mm.—when a second or third compression is made than when the first one was made only a few seconds before. He attributes this to the readjustment of the artery itself, as well as to the tissue and liquids surrounding it, to the new conditions under which the pressure has placed them. However, these slight changes are negligible in optometric work. They have been found practically the same in both fat and lean arms. They apply to both systolic and diastolic pressures.

Physiological Factors Influencing Blood-pressure

The various influences which might affect blood-pressure physiologically were partially enumerated in a previous chapter (Arterial Pathology), and may well be mentioned again, in addition to others. They consist chiefly of age, sex, time of day, position, state of mind (psychic), amount of liquids or solids ingested, heat, cold and exercise.

Age. In the chapter on Arterial Pathology it was suggested that probably the factor exercising the most invariable influence upon blood-pressure is age. There it was pointed out how sustained tension eventually terminates in tissue changes (hypertrophy), and what is true of sustained tension is also true, though to a lesser degree, of intermittent tension, *i.e.*, constant changes from relaxation to tension, and *vice versa*. Owing to the many factors cited, the arterial system is constantly subject to change throughout the entire life of the individual, a fact which is peculiarly true during the present age. It is natural that there should result an ultimate

degeneration in arterial tissue and a consequent resistance to influences which previously were met with acquiescence.

The resistance is evidenced by an elevation in the blood-pressure of the individual, concomitant with the passing years. There is a gradual rise, barring pathological influences, until 145 to 150 mm. is reached, after middle life. The following table illustrates the comparative changes which take place:

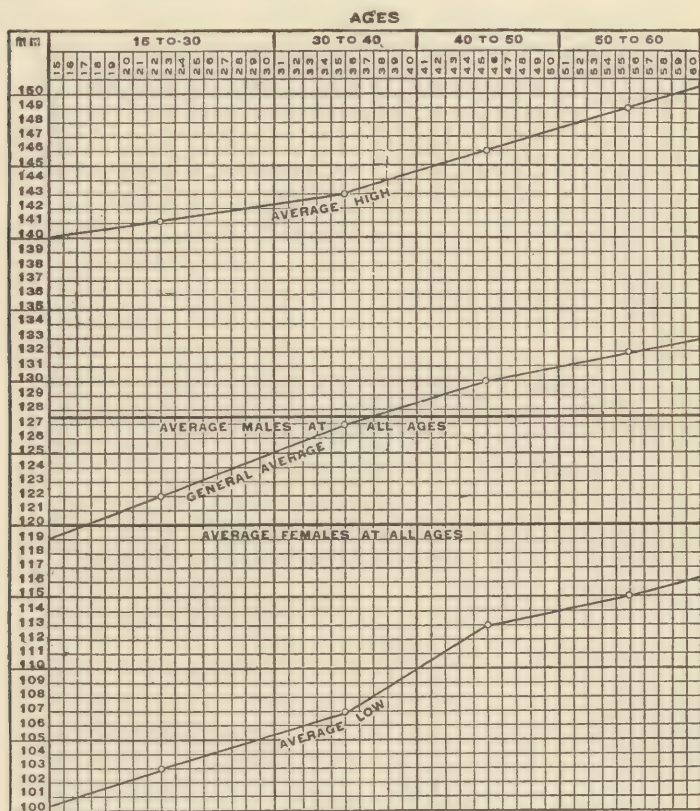


Fig 13

Chart Showing the Normal Limits of Variation in Systolic Blood-Pressure.
(Warfield, Woley)

It has been suggested that an excellent method of estimating the normal pressure for individuals over twenty years of age is to take 120 mm. as a basis, and add 1 mm. for each two years of the individual's age above twenty years. For example, one thirty years old would, theoretically, have a pressure of $120 + (10 \div 2) = 125$ mm. One fifty years old would have a pressure of $120 + (30 \div 2) = 135$ mm., and so on. The usual allowance of 5-10 mm. for the female sex should be made.

Of course, as previously stated, the pressure can vary rather widely in the individual and still remain within normal limits; therefore, this table cannot be accepted without qualification. However, experience and an intelligent understanding of the many factors involved enable the operator to differentiate between normal and abnormal conditions in the majority of cases, regardless of age.

Sex. Most authors seem to have arrived at the conclusion that blood-pressure in women may be expected to be 5 to 10 mm. lower than in men. The writer has not found this to be the case (see Chapter 14), but he acknowledges the more authoritative statements of those having greater experience in this matter.

Time. Blood-pressure is lowest in the early hours of the morning, gradually rising until late in the afternoon or evening. For example, a hard-working business man may be expected to have a higher pressure in the afternoon after a strenuous day than in the morning before tension has begun.

Position. According to Hirschfelder, "Erlanger and Hooker have shown that the minimal pressure rises considerably and the pulse pressure always decreases upon standing after having lain down. The pulse-rate increases accordingly. They have shown that these effects are

entirely due to the rule of gravity." The writer has found a difference of approximately 5 mm. in both systolic and diastolic pressures between a sitting and a standing posture, the latter giving the higher reading.

Psychic. The desirability of having the patient in a calm state of mind when taking the blood-pressure has already been pointed out. When there is failure to achieve this end, it is not at all uncommon to find, upon subsequent occasions, that the first examination revealed too high a reading. Excitement, anger, pain, worry and similar emotions tend to raise the pressure. Fear probably has an effect, but the writer has been unable to find reference to it in the medical literature at hand.

Eating and Drinking. These tend to raise the pressure several millimeters, perhaps because of increased mechanical tension, but more probably because of the increased heart action which the act of eating arouses.

Temperature. Heat exerts a depressing influence upon blood-pressure because of its effect upon the vasodilator system, while cold elevates the pressure through its effect upon the vasoconstrictor nerves.

Exercise. When extensive muscular action takes place, blood-pressure rises in proportion. After fatigue sets in, it tends to fall. If the exercise ceases before fatigue takes place, the pressure should fall more or less rapidly, and soon assume normality.

Normal Relationship Between Systolic and Diastolic Pressures

The arterial pressure-variations under pathological conditions have already been dealt upon briefly. It was stated that the systolic pressure in the adult could range from 100 to 150 mm. and still remain within normal

limits. This is true if the diastolic pressure varies more or less proportionately; otherwise, while the systolic reading may be normal, an abnormal diastolic registration might indicate a very grave state of health.

The normal relationship between the two pressures is approximately as 3:2; that is, a systolic pressure of 120 mm. should be accompanied by a diastolic pressure of about 80 mm. A higher pressure, say 135 mm. systolic, should have about 90 mm. diastolic.

This ratio holds good as long as the systolic pressure varies between 100 mm. and 150 mm. and the diastolic between 70 mm. and 100 mm. When either pressure escapes these limits, or the two are not maintained in approximately this ratio within the limits, there is reason to suspect pathological conditions.

For example, as pointed out under Aortic Regurgitation, a systolic pressure of 130 mm. combined with a diastolic pressure of 65 mm. may indicate an abnormal condition. Likewise, a systolic pressure of 115 mm., combined with a diastolic pressure of 95 mm., points to faulty conditions—a weak heart or a tubercular condition or both. The same disease might be expected if 140 mm. represented the systolic pressure and 105 or 110 mm. the diastolic. Yet 140 mm. is easily within normal limits.

While it has been stated that 100 mm. is the lowest systolic pressure that can be accepted as within normal limits, the writer wishes to emphasize that in optometric work 110 mm. really represents the lowest figure compatible with confidence on the part of the operator. Any case (of twenty years or more) possessing a lower systolic pressure than this should receive a most guarded prognosis and be carefully watched. It is among this class

of patients that the most discouraging disappointments occur, although it is perhaps equally true that frequently the correction of a slight ametropia yields brilliant results.

The reason for this is essentially that when the blood-pressure is represented by no higher figure, the circulation of the blood is evidently poor and there is a tendency toward the existence of the arterial and venous stasis previously suggested. The waste products of katabolism are, therefore, not removed with sufficient rapidity, and symptoms of ocular strain result.

If these symptoms arise entirely from the poor circulation, correction of refractive and muscular errors probably will not result in much improvement. On the other hand, if this circulation is sufficient for the normal demands of such an individual, but there exists an ocular defect which produces an unbalanced metabolism, the neutralization or removal of the defect may yield astonishingly satisfactory results. This is probably a partial explanation of why weak glasses often are so gratefully accepted.

Coincident with such a condition of the systolic pressure, there is likely to exist a destruction of the normal relationship between the systolic and diastolic pressures—the 3:2 ratio—in that the latter will exceed its normal proportion, giving a pulse pressure lower than is required to maintain a condition of health and comfort.

Pulse Pressure

As previously stated, the pulse pressure consists of the difference between the systolic pressure and the diastolic pressure. In the example of the pressures in aortic regurgitation, the pulse pressure is then the

difference between the systolic pressure—130 mm.—and the diastolic pressure—65 mm.—and is therefore 65 mm. In the same paragraph, the second example exhibits a pulse pressure of 20 mm. and the third a pulse pressure of 30 or 35 mm.

It has been rather definitely determined that the limits of the normal range of the pulse pressure are 25 to 50 mm. Less than 25 mm. is indicative of a weak heart, while more than 50 mm. indicates an unbalanced condition due to toxic or mechanical anomalies; for example, high systolic and normal or low diastolic pressures, resulting from renal or vascular diseases, is an illustration of high pulse pressure due to toxic causes, and normal or high systolic and low diastolic, due to aortic regurgitation or to arteries in which a persistent high pressure has materially widened the lumen, may be accepted as illustrations of mechanical causes.

So when the pulse pressure escapes from the limitations imposed it may be taken for granted that something is wrong. Although the optometrist is more concerned with this fact than he is with *what* is wrong, an intelligent understanding of such cases is advisable. It should always be borne in mind that the systolic pressure represents the full power of the heart that is being exerted during systole, that the diastolic pressure really represents the peripheral resistance and that the pulse pressure represents that part of the cardiac energy which is rendered potential in the walls of the arteries and which serves the purpose of driving the blood onward during diastole, the moment during which the heart rests. This latter pressure has been pertinently called the *head of pressure*.

These facts are offered that it may be clearly understood how important it is to ascertain both systolic and diastolic pressures. However, it must be conceded that the determination of the second pressure is not nearly as important to the optometrist as to the physician. The systolic pressure is the one which merits paramount consideration, as it more closely and accurately indicates departures from normal which seriously concern ocular welfare or cause the simulation of ocular strain.

Instruments

Much has been written about the various instruments used in estimating blood-pressure. Careful consideration of a somewhat extensive literature upon the subject leads to the conclusion that all instruments other than those of the mercury and aneroid types may be eliminated. These two types have stood the test of much use and may be regarded as accurate.

The relative merits of mercury and aneroid sphygmomanometers are, from a scientific viewpoint, about on a par, in the writer's opinion. The advocates of the mercury instrument argue that there is no metal in the instrument to become fatigued and therefore require adjustments, an argument of somewhat doubtful value.

Those who favor the aneroid type are not slow to point out that the inertia of mercury militates against its value in blood-pressure work, the point being that mercury requires 1.5 seconds to recover itself, while during the same period the heart beats 1.75 times. The action of the heart (and, of course, of the blood column) and the mercury column are therefore not synchronous, the result being that the fluctuation of the mercury is not sufficiently great to be plainly visible.

It is somewhat illuminating to note that the exponents of one type and the disparagers of the other are chiefly the inventors and manufacturers of the one they advocate. Those who are without interest in the matter, scientists, etc., have found both types accurate and satisfactory *from a scientific viewpoint*. In this respect, the faults of one are quite paralleled by the faults of the other.

From a practical viewpoint, however, there is no question but that the aneroid type is the most satisfactory. It is neat, compact, adjustable to any position the patient may take, owing to the hook by means of which it may be fastened to the armlet, is accurate to a remarkable degree, and is easily adjusted should it become injured; in this respect it differs materially from the mercury instrument, since the latter is more susceptible to breakage or loss of parts, the replacing of which may prove greatly inconvenient, tedious and expensive. Furthermore, the fluctuation of the hand upon the dial is plainly evident and registers the slightest action of the heart, a feature of inestimable importance, in the writer's opinion, as it yields information obtainable in no other way by the optometrist.

On the other hand, the mercury type must be carefully placed upon a table, and frequently in a position which makes the reading awkward; the action of the heart is not ascertainable by the movement of the column, and the glass tube may easily be broken or the mercury spilled.

In optometric practice, the aneroid instrument is undoubtedly the best. Of the several kinds on the market, the most prominent medical writers see fit to speak highly of only one, the Tycos.¹ Hirschfelder,

1. Manufactured by the Taylor Instrument Companies of Rochester, New York, U. S. A.

formerly of Johns Hopkins, in his monumental work "Diseases of the Heart and Aorta," says, "The writer has found instruments of the Tycos construction entirely free from variation for over two years, and still longer periods for absolute accuracy are on record."

Warfield says, "An instrument devised by Dr. Rogers (the 'Tycos') has met with considerable popularity. Contrary to the general belief, this is not an instrument which operates with a spring and lever. The instrument is composed essentially of two metal discs carefully ground and attached at their circumferences to the metal casing below the dial. There is an air chamber between these discs through the center of which air is forced by the syringe bulb. When air is forced into the space between these two discs, they are forced apart to a very slight extent; with the highest pressures only 2-3 mm. of bulging occurs. From data gathered after extensive use for two years, these discs were not found to have sprung. A lever attached to a cog, which in turn is attached to the dial needle, magnifies to an enormous extent the slightest expansion of the discs. Every dial is hand-made and every division is actually determined by using a U. S. government mercury manometer of standard type. No two dials therefore are alike in the spacing of the divisions of the scale, but every one is calibrated as an individual instrument. There is no doubt in the author's mind that for the general practitioner the instrument has some advantages over the mercury instruments. It reveals the slightest irregularity in force of the heart-beat. The oscillation of the dial needle is more accurately followed by the eye than is that of the column of mercury. The needle passes directly over the divisions of the scale, while with usual mercury instruments the scale is an appreciable distance (sometimes .5 cm.) from the

column of mercury at the side. The diastolic pressure is more easily read on the 'Tycos.' It is where the maximum oscillation of the needle occurs as the pressure is



Fig. 14

Tycos Sphygmomanometer

(Kindness of Taylor Instrument Cos.)

released from the cuff. Other firms are making instruments something like 'Tycos' in appearance. Possibly they are as good. There is this much to be said: no instrument using a spring as resistance to measure pressure can be recommended."

The writer of this work uses the Tycos and has found it eminently satisfactory. The self-verifying feature of this instrument gives it a value not possessed

by any other type of aneroid manometer. When no pressure is exerted and the hand rests *within* the limits of the oval zero, the instrument is correct and the reading may be depended upon. If the hand rests *without* the zero, the instrument is wrong, and this fact may be ascertained at a glance. Other aneroid instruments have a movable dial which can be set so as to accord with the hand when it comes to rest. It can readily be seen that with such an instrument any error that may exist in the metallic chambers is simply covered up, but not remedied. The starting

point might be all right, but, except by comparison with other instruments of known accuracy, it is impossible to tell whether readings beyond the starting point are correct.

The Tycos is used by every physician he has questioned in regard to sphygmomanometers. It is perhaps a trifle more expensive than some other instruments, but the value is there, and in a work of such importance as sphygmomanometry the best instrument should be used.

The manufacturers of this instrument also make a larger size of the same type (Fig. 15). It has an eight-inch face which may be read at fifteen feet. The eight-inch base upon which it rests may be placed upon a table or against the wall, there being an arrangement by means of which it can be hung upon screws. For demonstration purposes it is without an equal, and even in office work it has marked advantages. For general use, however, the smaller instrument is the more practical.



Fig. 15

Large Office Pattern Tycos Sphygmomanometer

SECTION TWO

THE RELATIONSHIP BETWEEN GENERAL AND OCULAR PATHOLOGY

Foreword

The essential purpose in presenting this Section is not so much to acquaint the student with the ocular diseases themselves as it is to point out their etiology, particularly their tendency to have the point of orientation in parts of the body other than the eyes.

For this reason the stereotyped list of ocular diseases usually found in ophthalmological and optometrical literature is not fully enumerated here, but rather is there given those pathologic disturbances of the general system which eventually terminate in ocular pathology if allowed to progress unchecked.

The importance of presenting this hitherto little considered subject—unconsidered in its present aspect, at least, in optometric literature—is obvious to the student and practitioner. As pointed out in the preceding pages, preventive measures are vastly more important than corrective ones and probably more efficacious in the majority of cases. It is easier to prevent the destruction of tissue than it is to repair it after destruction. The latter may be impossible.

It follows, then, that it is better to remove the cause of, say, optic neuritis, choroiditis, retinitis or cataract than it is to send such cases to the physician *after* the lesion has taken place.

That the ophthalmoscope is almost totally inadequate for this purpose is so apparent that logical argument is prohibited. This instrument, wonderful enough for its purpose, usually pictures an event that has already taken place, and the prognosis that can be based upon the information it reveals is more often a gloomy one than otherwise. In other words, it shows the examiner *destroyed tissue* or tissue in the process of being destroyed, and we all know that the prospects of recovery when the delicate ocular tissues are in either stage are very unfavorable.

Of far more importance, then, is it to discover the disease threatening invasion before it has had an opportunity to bring about ocular lesions or cerebral lesions affecting the ocular economy. This constitutes true preventive optometry.

The possibility of doing this has been made clear where it was stated that a great percentage of ocular diseases—particularly the intraocular varieties—are due to general diseases, and that of those which affect the eyes the majority register an abnormal sphygmomanometric reading. Even at the risk of repetition, this point should be emphasized.

With this thought in mind, the following partial list of diseases which affect the eye has been compiled and is here presented. Whenever it has been possible to gain definite information to that effect, it is stated just what state of blood-pressure can be expected in each disease, but it should be pointed out that medical literature is still undeveloped in this respect, and frequently the results

given by medical writers are contradictory or frankly incomplete or uncertain.

It is, of course, virtually impossible for an optometrist independently to discover the relation between blood-pressure and diseases foreign to his practice, owing to both paucity of material and lack of medical education. On the other hand, neither is it particularly desirable that he do so, since scientific research along such lines is more or less outside his province. The same amount of energy applied to the study of the relation between ocular functions and the blood-pressure would be productive of results far more important to optometric science.

CHAPTER 6.

DISEASES OF THE CIRCULATORY SYSTEM

As has already been pointed out in a preceding chapter, the direct or indirect effect of heart disease upon the ocular economy appears to be comparatively slight. Nevertheless, there is an occasional connection between cardiac and ocular affections.

Endocarditis, for example, may be cited. In this disease, which is an inflammation of the transparent lining of the heart, that portion of the membrane which covers the valves is said invariably to be affected first. The membrane becomes granular, the tissues destroyed and finally broken off and carried in the blood stream as emboli.

Embolism of the ocular vessels occurs in this manner. This affection takes place by there becoming lodged in the artery a clot or vegetation which has been detached from a diseased valve of the left side of the heart. Indeed, this condition has been spoken of as pathognomonic of cardiac disease, but it is true that it can be caused by emboli from other parts of the circulatory system. Parsons¹ attributes it to mitral stenosis which would result from the diseased valve, but also suggests that in reality the condition is often thrombotic, being due to the endarteritis of nephritis when the already narrowed lumen of the vessel becomes occluded.

1. Diseases of the Eye, page 378

When the embolus becomes lodged in the choroidal vessel, the condition appears to be of minor consequence—unless the obstruction is of an infectious nature—owing to the numerous anastomoses of these vessels. Knies¹ describes the condition as follows:

“A choroidal embolus looks like a chorioretinitis patch. It forms a whitish, somewhat prominent spot with indistinct borders, over which the retinal vessels pass or in which they disappear. In the latter localities there is considerable opacity of the innermost layers of the retina. The visual disturbance is essentially a scotoma corresponding to the situation of the embolus. After a time the opacity disappears, and more or less atrophy and irregular pigmentation of the choroid are left. Vision may be entirely restored. Such conditions are usually seen accidentally (because the subjective symptoms are slight), especially during convalescence from certain infectious diseases (small-pox, typhoid fever). Sometimes several foci develop at the same time or successively.”

Embolism of the retinal arteries is of far greater moment. To quote Parsons² upon this subject:

“Embolism of the central artery of the retina causes sudden and complete retinal anemia. The eye, usually the left, becomes suddenly quite blind. Examination of the fundus reveals a very typical picture. The larger arteries are reduced to threads, the smaller ones are invisible. The veins are little altered except on the disc, where they are contracted. Within a few hours the retina loses its transparency, becoming opaque milky-white, especially in the neighborhood of the disc and macula. Owing to the opacity of the retina, the outlines

1. The Eye in General Disease.

2. Diseases of the Eye.

of the disc, which is abnormally pale, are obscured. At the fovea centralis, where the retina is extremely thin, the red reflex from the choroid is visible. It appears as a round 'cherry-red' spot, presenting a strong contrast to the cloudy white background. The peculiar tint of the spot is due to this contrast. In the majority of cases there is no hemorrhage here, as was once thought, though hemorrhages here and in the immediate neighborhood do occur rarely. The contrast sometimes brings into relief minute blood-vessels near the macula which are otherwise invisible.

"Sometimes the obstruction to the blood flow is not complete, or the flow may be partially restored in the course of a few days. Another peculiar phenomenon may be observed or may be induced by gentle pressure upon the globe. In some of the vessels, usually veins, the column of blood is broken up into two red beads separated by clear interspaces. The beads move in a jerky fashion through the vessels, sometimes in the normal direction of the blood flow, sometimes in the opposite direction. If the veins are easily emptied of blood or arterial pulsation is produced by slight pressure on the eye-ball, it is evidence of incomplete blockage.

"The retinal edema or possibly coagulation necrosis takes several weeks to clear up. The membrane regains its transparency, but is completely atrophic. The vessels are contracted or reduced to white threads. The disc is atrophic. If there have been hemorrhages, spots of degeneration replace them, and cholesterol crystals and pigmented spots may be seen in the papillo-macular region.

"In some cases a certain type of central vision persists in spite of apparent complete occlusion of the central

artery. It is due to the presence of cilioretinal arteries which, when present, always supply the macular region and naturally escape occlusion. The remainder of the field of vision is lost.

"In other cases the embolism is arrested in a branch of the central artery. The area supplied by this branch is then affected alone. In the early stages the corresponding scotoma is usually somewhat indefinite, but later it settles down to a permanent sector-shaped defect.

"When perception of light is lost, the pupil is large and the direct reaction to light fails. In some cases faint perception of light may be regained in the temporal region of the field: it is possibly due to partial establishment of collateral circulation. The intraocular tension is normal, as might be expected."

Wood, in the American Encyclopedia of Ophthalmology, p. 4288, states that the cause of embolism of the central retinal artery is to be sought in lesions of the heart and arteries. He mentions the 125 cases tabulated by Fischer in which seventy per cent showed valvular lesions, recent endocarditis, arteriosclerosis, syphilitic endarteritis, or aneurysm. No adequate cause could be found for the other thirty per cent, but cardiac or vascular lesions were thought to account for half of them.

The author recently had a patient so afflicted. A young woman of about thirty came to him with 20-120 vision in the right eye, five months after her physician, an ophthalmologist, had stated she would never recover vision in that eye. The case had then been under his care for a month. By dimming the vision of the left eye, which was normal, with a dense red glass, it was possible to produce an intermittent diplopia. Working from this rather insecure basis with prisms, a course of exercises

was given which, in about four weeks, gave her 15-45 vision of a transient character. A more or less permanent diplopia was also present.

Notwithstanding this encouraging progress, it is doubtful whether there will be further improvement (everything except home treatment, which consisted of the application, alternatively, of hot and cold water, and of a gentle local massage while in a reclining position, was temporarily discontinued a month ago, owing to her inability to leave her work). The vision is evidently of peripheral character and is due not to revivification of retinal elements affected by the discontinued blood supply so much as to education of unaffected areas. Since the eye is now turned out to a perceptible extent, it is quite probable that a false macula will be created.

The fundus has a normal appearance except for the atrophic nerve-head and vessels reduced in caliber. There is also a very faint white border to one of the arteries. A cilioretinal artery is present, which may account for the partial vision. The pupil does not react to light directly applied, tho it does contract when light is thrown into the other eye and when an accommodative effort is undertaken.

(Since the above was written, about three or four months ago, the young woman has been observed several times. Somewhat to the writer's surprise, there has been a more or less constant improvement in her ocular condition. The vision has improved to 15-30, the pupil is contracting more and more to direct light stimulus and the patient finds the eye less and less uncomfortable. The answer is found in the development of the cilioretinal artery and veins. These vessels are fully double the width they were in the beginning, and that area of the nerve which

surrounds their exit is very perceptibly flushed. The rest of the nerve is atrophic. The final outcome of the case remains to be observed.)

CONDITION OF BLOOD-PRESSURE. There appears to be no specially characteristic blood-pressure symptoms of the diseases of the valves of the heart which could cause the above-described ocular condition. It is quite probable, however, that the disease which causes the cardiac lesions (syphilis, nephritis, etc.) will present evidence of its presence, and it follows that treatment of the cause will modify the possibility of the effect mentioned being produced.

Aortic Regurgitation or Insufficiency frequently causes the ocular phenomenon of arterial pulsation. The condition arises from the lowering of the arterial tension during diastole, caused by the reflux of a great volume of blood through the defective valves of the heart.

Under normal conditions, the lowest pressure within the retinal arteries, which naturally occurs during the moment of diastole, is at least equal to and perhaps greater than the normal intraocular tension. It is apparent, then, that at no time will the arteries decrease in volume if all other conditions are normal. Neither will they increase perceptibly, since to do so it would be necessary that they either compress into a smaller volume the contents of the globe or expand its walls. Neither feat is practicable, so the volume of the ocular arterial system therefore remains practically the same.

However, when the arterial tension falls below the intraocular tension, it is natural that conditions are changed. If the highest tension of the former became less than the lowest tension of the latter, complete retinal anemia would take place, but it is seldom that this

obtains for any great length of time, if, indeed, it ever does occur at all.

Nevertheless, it not infrequently occurs that the tension of the globe is midway between the highest and lowest tension of the artery, with the result that with each heart-beat the artery visibly expands and exceeds the tension of the globe, only to contract and become noticeably narrower when the wave of pressure passes. When sufficient blood has escaped from the eye to re-establish the balanced relationship of the two pressures, the vessel ceases to decrease in volume and remains stationary until again expanded by a new influx of blood.

When this margin of safety is dangerously small, it is not improbable that there are occasions when the highest arterial tension is not equal to the intraocular tension, and the patient complains of the so-called "black spells," when everything goes dark. Vision is re-established when the local pressure increases. Not only has arterial pulsation been observed in aortic regurgitation, but so also have alternate reddening and pallor of the optic papilla.

It is very probable that these conditions arouse the symptoms of ocular asthenopia. When the constant accumulation of the toxic products of ocular metabolism is accompanied by a depleted blood current, it is most natural that the symptoms of strain and over-exertion appear. This subject is more fully presented under Anemia.

CONDITION OF BLOOD-PRESSURE. This form of heart disease has a very characteristic blood-pressure presentation. In consequence of the reflux of blood through the defective valves, it becomes necessary that the heart force into the arterial system a greater quantity

of blood than would otherwise be necessary. This naturally results in a pronounced rise in pressure for the moment, constituting the systolic pressure. Owing now to the fact that the blood is rapidly escaping by two means of egress instead of one—by both the capillary system and the insufficient valves of the heart—there occurs a rapid and extended *fall* in the arterial pressure, the lowest point of which constitutes the so-called *pulse pressure*, and its extent, in relation to the systolic and the diastolic pressures—the highest and the lowest—is of the greatest diagnostic importance.

For example, assuming the normal relationship to be

Systolic Pressure.....	120
Diastolic “.....	80
Pulse.....	40

it is found that the pulse pressure is equal to one-half the diastolic and one-third the systolic. Suppose a case is encountered in which the following relationship exists:

Systolic Pressure.....	130
Diastolic “.....	65
Pulse.....	65

It is here observed that the pulse pressure is one-half the systolic and equal to the diastolic pressure. It becomes at once clear that the normal relationship is destroyed, and that something is wrong.

The visible pulsation of the artery alone has been said to be always pathological.¹ When taken in conjunction with an abnormal state of blood-pressure as mentioned, there can be no question of the existence of diseased conditions. The case is one for the physician.

1. Parsons' Diseases of the Eye, page 150.

Dilatation of Heart has been observed by Knapp coincidently with general cyanosis, aneurysmal murmurs in several parts of the body and excessive hyperemia of the retina, with thick and sinuous arteries and veins.

CONDITION OF BLOOD-PRESSURE. As the condition is due to a weakness of the heart muscle, it is probable that there is present a normal diastolic pressure with a low systolic pressure and a correspondingly low pulse pressure.

Aneurysm of the Aorta¹ and other sections of the arterial system has been associated with ptosis, myosis and enophthalmos. It may give rise to retinal pulsation, optic neuritis and choked disk.

The latter occurs probably when the aneurysm is present in the orbit and the distended artery is involving the optic nerve.

When it exists in the cavernous sinus there may be affected the nerves which accompany the internal carotid artery in its passage through that channel—the oculomotor, abducens, trochlear and trigeminus. Aneurysm of the ophthalmic artery within the orbit has been reported several times. In one case,² the saccular extent of the artery was compared to an orange; in another,³ to a walnut, a more probable relation.

CONDITION OF BLOOD-PRESSURE. The author has been unable to find any reference to the state of blood-pressure in aneurysm. It probably has no characteristic pressure, but when the condition exists in one of the larger arteries and the distended portion accommodates a large quantity of blood, it seems not unreasonable to suppose

1. Knies, *Eye in Relation to General Diseases*, page 296.

2. *American Encyclopedia of Ophthalmology*, Vol. 1, page 461.

3. *Loc. cit.*

that the blood-pressures would be very much similar to those attending dilatation of the heart—low systolic, normal diastolic.

Arteriosclerosis is the characteristic disease of the arterial system. Constituting, as it does, a destruction of elasticity in a system whose chief attribute is the ability to contract and expand according to the needs of the surrounding tissue, it can readily be appreciated that the presence of this disease may lead to grave consequences.

The ocular manifestations of its presence are given as follows in the American Encyclopedia of Ophthalmology, Vol. I, page 614:

"1. Alterations in the course and caliber of the retinal arteries, manifesting themselves as: (a) undue tortuosity, which is not significant unless, to quote the words of Mr. Gunn, whose classification is followed, it is associated with other evidence of disease; (b) alterations in the size and breadth of the retinal arteries, presenting, as it were, a beaded appearance. 2. Alterations in the reflections from, and the translucency of, the walls of the retinal artery, manifesting themselves (a) in increased distinctness of the central light streak on the retinal vessel and an unusual light color of the entire breadth of the artery; (b) loss of translucency, so that it is impossible to see, as is possible in the normal state, through the artery an underlying vein at the point of crossing; (c) positive changes in the arterial walls, consisting of whitish stripes, indicating degeneration of the walls or infiltration of the perivascular lymph-sheaths (perivasculitis). 3. Alterations in the course and caliber of the veins, together with signs of mechanical pressure, manifesting themselves (a) in undue tortuosity, which, as in

the case of the arteries, is not significant except in the presence of other disease; (b) alternate contractions and dilatations; (c) and impeded venous circulation where a diseased artery crosses it. The last is a sign of the utmost importance. Ordinarily, as an artery crosses the vein, as it may be seen by an examination of the normal eye-ground, there is no sign of pressure, and the translucent artery permits a view of the vein beneath it. If the walls of the artery are thickened by disease, then it presses upon the vein, pushes it aside or directly contracts its caliber, so that beyond the point of crossing there is an ampulliform dilatation; (d) changes in the venous walls, precisely as they occur in the arteries, so that whitish stripes border the vessel, and are indications of degeneration in its walls. Often associated with this one may see varicosities. 4. Edema of the retina, manifesting itself as a grayish opacity, which may be present in the immediate neighborhood of the papilla, or in spots over the eye-ground and along the course of the vessels, looking like a fine gray haze, or in little fluffy islands far out in the periphery. 5. Hemorrhages, manifesting themselves as linear extravasations along the course of the vessels, roundish infiltrations scattered over the fundus, or sometimes in a drop-like form."

Warfield¹ emphasizes the value of the eye-ground phenomena in this disease and states that "long before there are any suggestive symptoms, changes can be seen in the blood-vessels of the retina, which, while not always diagnostic, at least call attention to a beginning chronic disease." He gives as the most important diagnostic symptom of all the indentation of the veins by stiffened arteries, a condition very frequently observed in optometric practice.

1. Arteriosclerosis, 135.

He states, furthermore, that every one having a pronounced arcus senilis, the whitish ring bordering the corneal margin, has arteriosclerosis, adding, however, that the converse is not necessarily true.

The general subjective symptoms given by him are gradual loss of acute vision, and attacks of momentary loss of vision. It is probable, however, that these symptoms arise from a complicating nephritis or syphilis. This would apply at least to the decrease in acute vision, according to the author's experience. Probably the transient loss of vision would arise from the contraction of a diseased central artery, as suggested by others.

As for the involvement of the retinal vessels in general arteriosclerosis, it is rather frequent, having been found twenty-four times in forty-four cases, by Raehlman.

GENERAL EFFECT

It is unnecessary to dwell in detail upon the effect of arteriosclerosis upon the general system. The tables previously given show its position in mortality reports. If the changes in the vessels that are brought about by other diseases—nephritis, syphilis, infectious embolism, etc.—could be legitimately designated as uncomplicated sclerosis, the percentage of deaths due to the disease would be enormously increased. For apoplexy, for instance, would then come under the heading of arteriosclerosis, this occurrence being due, usually, to the rupture of a diseased cranial artery.

But it would be impracticable to thus enlarge the province of the disease, as it would lead to endless complications and rob other diseases of their rightful importance. Therefore, it has been deemed proper, as stated previously, to give, in mortality tables, the final cause of

death and not that which may have been an inciting factor. For this reason, and justly so under the circumstances, arteriosclerosis in these reports does not loom high.

Nevertheless, if we disregard the etiology of the disease and consider only the condition itself, it will be found to exist in a very great proportion of cases and exert a very grave influence generally.

Warfield states "there may be dull headache that the accurate fitting of glasses does not alleviate." Vertigo, weariness from slight effort, tinnitus (ringing in ears), numbness of hands and feet, epistaxis, dyspepsia, insomnia, irritability, and intermittent limp are some of the general symptoms given. It will be observed that several of them are strikingly similar to the symptoms of ocular strain.

The graver effects of this condition are seen in the major organs of the body—the heart, kidneys, brain, spine, lungs and vasomotor system—for it appears to be true that disease of the arteries can affect these organs as well as be affected by them. For the purpose of arousing a proper realization of the effect of the disease in these organs, it suffices to say that its termination is fatal in many instances. Heart failure, apoplexy, auto-intoxication (constituting a vicious circle with kidney disease?), epileptic convulsions and pulmonary hemorrhages may be mentioned in this connection.

CONDITION OF BLOOD-PRESSURE. This symptom of arterial sclerosis is variable and depends upon whether (1) the condition is general, (2) localized, (3) where localized, and (4) whether other diseases complicate matters.

It appears that hypertension is not necessarily always the result of the disease, since hypotension also can take place.

Medical writers do not seem to be in exact agreement in the matter. For example, one states that arteriosclerosis is "a disease of the arterial system characterized by chronic degenerative changes in the walls of the arteries, causing a persistent and very high blood-pressure," and another says, "in this condition we find a permanently elevated blood-pressure," while a third—Warfield—advances the statement that "in arterial sclerosis, especially the diffuse 'senile' type, the blood-pressure is invariably low, and may be spoken of as hypotension."¹

It is very probable that these statements are all subject to qualifications. The first two writers very likely had in mind the fact that usually arteriosclerosis is associated with hypertension and, at the point quoted, did not feel called upon to differentiate between the sclerosis caused by high tension and the high tension caused by sclerosis. On the other hand, the latter writer probably referred to the idiopathic form of the disease in which there were no complications, just hardened arteries without nephritic or syphilitic causes.

The correctness of his surmise seems to be supported by Prof. Romberg's² conclusion that there is a high blood-pressure only in such cases of arteriosclerosis where there is a diseased condition of the kidneys. Lorand³ "especially insists on the fact that high blood-pressure is not a condition essential to arteriosclerosis."

1. Arteriosclerosis, page 90.

2. Old Age Deferred, A. Lorand, page 165.

3. Old Age Deferred, page 164.

Very likely there would be a complete agreement if the conclusions of each were analyzed and qualified. When a proposition is subject to as many complications as this one is, it would be impractical to elaborate upon and explain each statement made, since the didactic value of the whole would thereby be greatly lessened. Therefore, medical writers may seem to disagree, yet be in perfect accord concerning basic factors.

These factors have been enumerated and may now be considered.¹

GENERAL arteriosclerosis influences the blood-pressure according to the stage to which the disease has progressed. It appears that in the initial stages there is a hardening of the middle coats of the arteries and perhaps a narrowing of the lumen, owing to the thickening of either the middle coat or the inner coat or both. The hardening and thickening may both take place, and the result is a decrease in the volume of the system and also a decrease in its elasticity.

In this stage the arteries more nearly resemble a system of inelastic tubes, and the natural consequence is an elevated blood-pressure. Systolic pressure will be markedly high and diastolic pressure approximately normal, the combination naturally resulting in a high pulse-pressure. Example: systolic 180 mm.; diastolic 90 mm.; pulse-pressure 90 mm.

As the disease progresses, the coats of the arteries become weakened and distended, with a consequent widening of the lumen of the vessels. It is a natural sequence that the blood-pressure should fall. The sys-

1. The writer may be pardoned if he here appears inconsistent and relapses into apparent redundancy, when the reader is reminded that the condition of the arterial system forms the basis of sphygmomanometry. A thorough explanation of arterial phenomena is necessary for a correct understanding of the subject, and reiteration may profitably be risked.

tolic pressure will be low, the diastolic normal or low, and the pulse-pressure low.

LOCALIZED arteriosclerosis affects the blood-pressure according to the extent of the lesion and *where* located. If it is slight in area, there will be no perceptible effect; when it is extensive, the effect may be marked.

Peripheral sclerosis, for example, involves a vast network of vessels capable of accommodating a high proportion of the blood. When these vessels have their lumen narrowed and their walls hardened, their capacity is reduced and less liable to variations. The result is that they effectively prevent a normal outflow of blood under normal pressure, and therefore the fluid is "backed up" in the larger vessels. Under these circumstances the sphygmomanometric reading gives a high systolic pressure, a high diastolic pressure, and a normal or low pulse-pressure. If, however, this condition has lasted for some time, the vessels eventually are widened and weakened so that they do not present a normal resistance. This circumstance leads to a normal or low systolic and a low diastolic pressure. It is probably this condition Warfield had in mind when he stated that in arteriosclerosis the blood-pressure is invariably low, whereas the others evidently referred to the former one, in which the sclerosis was in the primary stage.

Sclerosis of the aorta and other large vessels presents a picture not greatly dissimilar to the one offered by a like condition of the peripheral vessels. As this portion of the system accommodates the immediate influx of blood from the heart, its inability to expand, when sclerosed, naturally throws a greater burden upon the peripheral vessels. The result is shown in a high systolic pressure and a moderately high diastolic pressure, the

latter being perhaps not as high as when the peripheral vessels were hardened and narrowed, since the normal peripheral vessels permit a normal escape of blood, more than when they are narrowed, and less than when they are permanently widened.

When the aorta has degenerated into the aneurysmal state and contains more than its normal quantity of blood, the systolic pressure may be low, the diastolic normal or high, and the pulse-pressure low.

OTHER DISEASES, of course, complicate matters. It has been said that the blood-pressure is always high if nephritis exists in conjunction with arteriosclerosis. It is conceivable that it may be low in spite of a general sclerosis which would ordinarily cause a high pressure, if anemia were present. The one offsets the other under such conditions. The same reasoning applies to other diseases affecting the blood-pressure. Those lowering the pressure tend to affect the elevated condition due to sclerosis and make still lower a depressed condition due to the same cause. Those elevating it act conversely.

CHAPTER 7.

DISEASES OF THE DIGESTIVE SYSTEM

It has been pointed out in the preceding pages that the influence of diseases of the digestive system upon the ocular system is slight, altho the converse is well known to be of paramount importance. When such influences are at work, the ocular effect is of a transitory nature in the vast majority of cases, consisting of temporary pains, principally in rotating the eyes; visual disturbances in the form of scintillating scotoma, and a repugnance to accommodative effort.

These symptoms are merely part of the general malaise which usually passes off when the toxic products of a deranged digestion are eliminated from the system. Should they persist, other pathologic conditions may be suspected. For example, two cases have been cited to the writer as the result of gastric disturbances, the one being a temporary change of one diopter in the astigmatism of one eye, and the other the spontaneous appearance of hyperphoria which gradually increased to five degrees and as gradually decreased until entirely eliminated. It is believed that if there were gastric disturbances associated with these phenomena, they were merely coincidental and not causative. The cause was probably syphilitic or nephritic, altho it is true that it may have been dental. It has frequently been reported that anomalies

of the ocular muscles have completely disappeared upon the removal of a decayed tooth, or the institution of other forms of hygiene in the oral cavity. This will subsequently receive the attention due to its importance.

Cancer, ulcer of the stomach and appendicitis, which appear with great frequency in vital statistics, seem to present no ocular symptoms of pronounced character, unless, indeed, sarcoma of the choroid could arise from the first mentioned. Neither does there seem to be a characteristic blood-pressure associated with them, tho it has been stated that a low pressure may be the result of cancer when the latter causes a wasting of the system.

The symptoms of these diseases may simulate the symptoms of eye-strain, and *vice versa*. The author has in mind a recent case—a woman—who was afflicted with sharp pains in the region of the appendix. These began several years ago and occurred at intervals of a month or so, becoming more frequent as time passed. Finally, an operation was performed and the appendix removed, with no resultant effect, apparently, as the pains continued and increased in frequency and intensity until they were practically a daily occurrence for the three months previous to the patient's appeal to the writer for aid.

The relating of her symptoms included no mention of these pains, the chief symptoms complained of being car-sickness and headache. Glasses correcting about three-quarters of a diopter of myopic astigmatism were given, and since that day she has had the pain above-mentioned only once. The nausea and headache, also, were relieved.

It is difficult to understand how such a pain could be part of the sequelae of eye-strain, unless it arose from the

presence in the intestine of undigested and putrefying food materials which were in such a state because of the reduced efficiency of the digestive apparatus, this, in turn, being due to the debilitating influence of an exhausting ocular strain. The chain of circumstances when thus traced is clear.

Diarrhea, it is conceivable, may have a like etiology. This condition, which may become more or less chronic and lead to death, has been associated with very pronounced phenomena. Immerman¹ has reported a case of blindness and subsequent atrophy of the optic nerve after very profuse diarrhea. The condition has also been known to give rise to xerosis of the conjunctiva and of the cornea, with a consequent ulceration. Imperfect movements of the lids have also been reported.

CONDITION OF BLOOD-PRESSURE. Owing to the loss of fluids, the disturbance presents a low blood-pressure. A continually lowering blood-pressure points to an early collapse.

Constipation certainly presents some of the symptoms of eye-strain, but whether or not it can cause even a passing ocular anomaly is a matter apparently not yet definitely determined.

The symptoms consist chiefly of a dull headache, hardly to be differentiated from a strictly ocular headache by any one except, perhaps, the sufferer, and a general mental and physical lassitude and dullness. Regarding the possibility of the condition more or less directly affecting the organ of vision, it does not seem altogether inconceivable that this could occur through auto-intoxication. There seems little doubt that failure to expel the

1. Knies, page 270. In the absence of more information concerning the case, it seems reasonable to assume that the actual cause was due to an associated disease which permitted a retrobulbar hemorrhage.

waste products of digestion results in their re-absorption into the system in the course of time. The action of these toxic substances upon the general system is so pronounced that there is no justification in supposing the ocular economy to be immune.

These thoughts are prompted by a mental review of two cases, husband and wife, so identically alike that in all the salient features they may be considered as one. If anything, the woman's was perhaps the more pronounced. Her history consisted of a general neurasthenia, with all that the condition entailed—headache, indigestion, constipation, a general run-down state, etc. She had visited one oculist after another, some of them excellent refractionists, and one optometrist other than the writer. The result was always indifferent; perhaps relief for a time, but an eventual relapse. Her refraction was gone over carefully and only a slight change was made in the lenses she was wearing. They consisted essentially of compound plus lenses, not exceeding one diopter in strength, with the axes of the half-diopter cylinders ten or fifteen degrees oblique.

Her report after two weeks was encouraging and her eyes were gone over again. While there was no change in their refraction, it was found that about one degree of esophoria had been uncovered(?). After this was verified at a later examination and the amount had increased to one and a half degrees, a prism, one degree base out, was given.

To make a long story short, her case was observed for several months during which time her *apparent* esophoria increased to five or six degrees, and it was gradually corrected to the extent of four degrees. At first, the relief was remarkable. The woman gained greatly in

weight and reported great encouragement. Later she had the usual relapse and was as bad as ever.

She next consulted another refractionist, who, I have learned from her husband, took off her prisms and substituted simpler lenses, with a result at least no worse than the one I obtained, altho hardly any better either, according to all reports.

The husband's case ran a parallel course, the *apparent* esophoria equalling that of the wife. He, also, was given lenses without prisms by the above mentioned refractionists, with similar indifferent results.

It should be mentioned that neither of these patients had pronounced ocular (local) symptoms of strain with their distance glasses. The local discomfort took place chiefly in reading, this act becoming intolerable after a few moments, regardless of lenses given. The ages of the patients were approximately forty-five, certainly the most difficult age at which to fit lenses, especially bifocal lenses. The three predominant features in their cases were the inability to use the eyes for close work, the appearance of an apparent esophoria, and a condition of chronic constipation. Neither of the two hardly ever had a normal movement of the bowels, artificial means being nearly always resorted to.

Is it possible that the toxic products re-introduced into the blood stream so affected the ocular system that they constituted an etiological factor in the creation of the apparent esophoria? Could these poisonous substances irritate the internal rectus muscle into spasmodic action, or inhibit the function of the external rectus so that it permitted an inward deviation of the eye? Again, how can be explained the apparent desire of the extrinsic muscles to always present an error of one or two degrees

in excess of the prismatic corrections the patients were wearing?

These questions cannot at this time be answered by the writer and he cannot remember having read attempts of other authors to explain this condition. Perhaps the peculiar combination above mentioned has not been observed by others, or if so, no conclusions were drawn as to their inter-relationship. That there is a recognized relationship between constipation and ocular diseases is evidenced by the following paragraph quoted from the American Encyclopedia of Ophthalmology, page 3268: "Constipation. This condition of the digestive apparatus is but one of the many symptoms related indirectly to a number of diseases of the eye—especially those of the uveal tract. Opacities in the vitreous is also one of these, and, in the treatment of the various forms of uveitis and its consequences, elimination from the body of morbid material is a most important matter. This subject will be taken up more fully under the caption of General Diseases in Their Relation to the Eye."

Unfortunately, the volume containing information concerning general diseases in their relation to the eye is not off the press at this writing, and therefore the information it contains is not available. It is apropos, however, to mention here the fact that de Schweinitz and Fife (*Ophthalmic Year Book*, 1909, page 184) apparently believe that altho no ocular disease is positively known to be due to intestinal auto-intoxication, and that laboratory examinations have failed to establish such a connection, an examination for intestinal toxins is advisable.

In the cases mentioned, an interesting and tenable hypothesis concerning the underlying cause of the pseudo-

esophoria could be built upon known facts relating to muscle anatomy and physiology. Tigerstedt¹ presents some very interesting information in this respect, but space will not permit its introduction here.

The straining incidental to this condition may be instrumental in bringing about ocular lesions, as is evidenced by the following paragraph from Knies²:

"Conjunctival hemorrhages occur not infrequently in old people with brittle vessels, from straining at stool. Hemorrhages into the retina or vitreous, or into the orbit, are much rarer. Such hemorrhages possess prognostic importance because, as a rule, *they are forerunners of cerebral apoplexy.*"

CONDITION OF BLOOD-PRESSURE. There seems to be no characteristic blood-pressure associated with this condition. It would appear that the re-absorption of toxic products should influence the vasomotor nerves in the usual manner of such substances, and it is very probable that it does, for, as Visscher³ states, the products of putrification have high pressor qualities, but it is possible the effect is so comparatively slight that it is apparent only to the physician who has observed a case daily for an extended period of time.

One is inclined to think there *must* be an effect upon the sensitive vasomotor nerves when it is realized that the same condition can influence cardiac action, as stated by Hirschfelder.⁴ Yet, W. H. Sheldon⁵ has said, "Headache and dizziness supposed to be due to high

1. Text Book of Human Physiology, chap. XV.

2. The Eye in Relation to Disease, page 270.

3. Blood-pressure in General Practice, Nicholson, page 84.

4. Diseases of Heart and Aorta, page 709.

5. Medical Record, December 31, 1910.

pressure, I have again and again seen disappear with *laxatives* and diet and *no change in blood-pressure.*"

It can at least be said that owing to both the toxic and mechanical features involved there is a tendency to elevated blood-pressure in constipation.

Dental Abnormalities have been suggested as causative factors in ocular disturbances. Muscular spasms and paralysis are very positively known to have been caused by defective teeth. Of this character, affections of the internal and external recti muscles and of the ciliary muscle probably predominate, disturbances of visual acuity being less frequent. Internal strabismus has been reported as relieved by extraction of decayed or impacted teeth. Likewise, amblyopia and amaurosis.

Knies has presented the most comprehensive collection of data in this respect, and the writer feels that although his work, "The Eye in General Diseases," is not a strictly modern one, it covers the subject to a degree not surpassed in more modern ophthalmological literature. He is quoted verbatim herewith¹:

"Apart from abscesses of the gums, especially of the so-called eye-teeth, which are occasionally mistaken for dacryocystitis (the reverse is also true in many cases), a definite relation is established mainly in reflex neurotic conditions (pupillary changes, paralyses, spasms, etc.).

"Conjunctivitis and phlyctenulae are said to result from teething, but the former are so frequent that this relationship is very uncertain. During the teething period, disturbances of function can only be recognized when they are very marked, and for this reason they are undoubtedly overlooked in the majority of cases.

“Numerous reports have been made concerning ocular affections and toothache in adults. Keratitis, iritis, phlyctenulae, glaucoma, paralyses, asthenopia, amblyopia without lesion, supraorbital neuralgia, exophthalmus, etc., are mentioned; restriction of accommodation is mentioned with special frequency. Schmidt (*Arch. f. Ophth.*, xiv, 1, p. 107) found it seventy-three times among ninety-two cases, either bilateral or unilateral (in the latter event, only on the side of the toothache); it was most frequent in youth and amounted to 5.0 D. or more. It rarely gave rise to subjective symptoms. Schmidt believes that it is due to reflex increase of pressure in the eye, analogous to the restriction of accommodation occasionally observed as a prodromal symptom of glaucoma. Pathological anatomy, however, teaches that the latter has a much more material local cause. When limitation of accommodation appears during toothache, it probably results simply from the lack of vigorous innervation on account of the distressing pain. Muscular insufficiency and diplopia, which are occasionally observed, may also be explained as paresis due to enfeebled innervation.

“On the other hand, spasm of accommodation has also been observed as a nervous symptom in toothache. I have seen nictitation disappear immediately after removal of a painful tooth. Gosselin makes a similar statement concerning supraorbital neuralgia. Neuralgias, particularly in the first and second branches of the trigeminus, infection of the eyes and epiphora may undoubtedly result from toothache. In view, however, of the very great frequency of toothache, the connection must at least be made probable by the fact that with cessation of the latter, or with removal of the diseased tooth, recovery or at least striking improvement of the neuralgia sets in.

A certain degree of relief of neuralgia is probably always associated with the removal of a diseased and painful tooth.

“Amblyopia and amaurosis as the result of disease of the teeth have been reported several times,—for example, by Lardier (*Rec. d'Ophth.*, 1875, p. 87), Gill (*Jahr. f. Aug.* 1872, p. 195). When the symptoms appear without any visible lesion, the connection may be granted. But positive ophthalmoscopic findings make this extremely suspicious and lead us to think of a common cause for the pain and visual disturbance.

“Hutchinson (*ib.*, 1885, p. 316) observed lagophthalmus from spasm of the levator palpebrae superioris during toothache. Blanc (*ib.*, 1871, p. 225) claims to have cured chronic ophthalmia(?) by extraction of a tooth. Brunschwig (*ib.*, 1887, p. 302) makes the same claim with regard to two causes of iritis with hypopyon. It is easy to understand the frequent development of abscesses in the lower lid as the result of abscesses around the teeth, especially of the so-called eye-tooth. Very rare is purulent inflammation of the orbit in caries of the upper teeth. Pagenstecher (*Jahr. f. Aug.* 1884, p. 620), Burnett (*ib.*, 1885, p. 16) and Vossius (*Arch. f. Ophth.* xxx, 3) have observed causes of this kind. Exophthalmus may also develop from serous infiltration of the orbital tissue in the vicinity of the inflammatory focus and it may disappear rapidly after removal of the tooth. Dimmer (*Wien. med. Woch.*, 1883, p. 299) observed metastatic choroiditis after extraction of a tooth; Sewill claims to have seen the development of cataract after this procedure. Neuschueler (*Jahr. f. Aug.* 1889, p. 403) reports the cure of toothache by means of prisms.

“On the other hand, it is to be noted that pain in the upper teeth on the same side is a frequent symptom of the

so-called ciliary pains of keratitis, but particularly of iritis and cyclitis. Less frequently is there pain in the teeth of the lower jaw on the same side; these are cases of irradiation of pain to adjacent nerve-twigs. Neuralgic toothache may be the prodromal sign of glaucoma."

A. Morgan MacWhinnie has reported a case (New York Medical Journal) in which reading was accomplished with great difficulty, being impossible after fifteen or twenty minutes. The symptoms consisted of severe ocular pains. An X-ray examination demonstrated an abscess at the root of the left cuspid, and the subsequent removal of 1.5 c. c. of pus completely relieved the trouble. The same author has reported ocular hemorrhages and temporary blindness.

Impacted teeth have been known to have caused somewhat similar disturbances.

Ocular disturbances similar to those mentioned could be recited *ad infinitum*, but it is beyond the province of this work to cover the subject completely and in detail.

CONDITION OF BLOOD-PRESSURE. The writer has been unable to find any reference in literature to the relation between dental abnormalities and the associated blood-pressure, and personal inquiries have yielded no more definite information.

It seems commonly agreed among dental surgeons, however, that a psychic effect could bring about a pressure change. The intense pain must exert an influence, but the character of such influence would depend perhaps upon the individual affected. In one of great resistance it might bring about a tenseness, resulting in an elevated

pressure. In a more yielding individual, there could occur a collapse, with a consequent depressed arterial tension.

An abscess constantly giving off quantities of pus, which is swallowed, would naturally tend to poison the system and effect lesions, a sequela of which would be an altered arterial tension. It would be difficult, not to say impossible, to determine what character of change would be effected. It is not likely it would be pronounced or permanent.

HEPATIC DISEASES

The connection between liver and ocular disturbances appears to be rather obscure and more or less negligible.

Jaundice may be mentioned in this connection, since subjective yellow vision has been observed in association with it. Knies¹ thinks the ocular symptom is due to the action of ptomaine, and that the non-production of ptomaines in some cases accounts for the absence of the yellow vision.

Hemeralopia—night blindness—has also been found associated with liver diseases. Likewise, xerosis of the cornea.

Retinal hemorrhages have been mentioned as occurring in the terminal stage of acute yellow atrophy of the liver. They are due to the septic poisoning.

Hirschberg has suggested that bilateral retinitis may arise from hepatic diseases. Xanthelasma due to the same cause has been reported.

1. Knies, page 271.

Knies¹ questions Landolt's theory that there is a connection between

Cirrhosis of the Liver and retinitis pigmentosa. He points out that the rarity of their association and their divergent etiology render such a hypothesis untenable. At the present time the cause of retinitis pigmentosa is unknown.

CONDITION OF BLOOD-PRESSURE. In jaundice it is believed the blood-pressure is inclined to be low—around 100–110. There appears to be little said upon the subject. In cirrhosis of the liver the pressure is thought to be high.

1. Knies, page 272.

CHAPTER 8.

DISEASES OF THE URINARY SYSTEM

This subject is rich in facts, probabilities and possibilities. No portion of the field of pathology exercises, in the aggregate, so great an influence upon the ocular economy or intrudes so frequently into optometric practice. Few, if any, diseases so potently influence adult life, as reference to the tables given in Chapter I will testify, if it is conceded that a large percentage of diseases of the vascular system are caused by renal diseases.

Albuminuria—**BRIGHT'S DISEASE**—**NEPHRITIS**, sometimes resulting in the classic stellate picture of albuminuric retinitis and lesser forms of fundus abnormalities, can be presented as the dominating factor in this study. Its ocular manifestations range from lesions seemingly without effect subjectively, or scarcely discernible objectively, to partial or complete blindness, and to the spectacular appearance presented by thrombosis of the central retinal vein.

Included in the list of ocular affections associated with the disease may be mentioned albuminuric retinitis, hemorrhagic retinitis, paralyses of the ocular muscles, iritis, cyclitis, cataract, choroiditis, optic neuritis, atrophy of the optic nerve, vitreous opacities and liquification, retrobulbar neuritis, local, as well as general, arteriosclero-

sis and phlebosclerosis, detachment of the retina, hemorrhagic glaucoma, embolism of the central retinal artery, and subconjunctival hemorrhage.

The subjective symptoms may be photophobia, blurred vision, painful vision, headache (often matitudinal), nausea, dizziness, vomiting and other gastric and intestinal disturbances.

The frequency of kidney disease has already been indicated. The frequency of ocular symptoms of the disease may be understood by the following quotation from the American Encyclopedia of Ophthalmology, Vol. I, page 208:

"Of 935 cases of kidney disease, tabulated by Groenauw and Uhthoff, albuminuric retinitis was present in 209, or 22.4 per cent. Wagner met it in six per cent of his cases, while in Galezowski's cases thirty-one per cent showed albuminuric retinitis. While the disease has been observed a few times in children, as a rule the patients are over forty years of age, and the majority are between forty and fifty years old. Men are more frequently affected than women, in the proportion of about seven to three. As regards the form of kidney lesion present in albuminuric retinitis, the small contracted kidney is the most frequent; chronic diffuse parenchymatous nephritis (large white kidney) forms a close second; the nephritis of scarlatina is third; and the least frequent are the rare cases of amyloid degeneration. As a rule, both eyes are involved in the retinal changes, and it rarely happens that the second eye remains entirely well for the period of a year.

"According to Porter, in the majority of cases of renal disease there is no disease of the retina; and a majority of renal cases showing retinal changes also show changes in

the blood-vessels. He concludes that the eye disease does not depend so much on the existence of the renal affection as on the fact that the vessels are diseased. Almost without exception the cases of albuminuric retinitis which he observed were found in syphilitics. Owing to vascular changes incident to syphilis, hemorrhages and fatty degenerations are to be expected in the retina. In nearly four thousand post-mortem examinations he has never seen one-sided nephritis. Some foreign authors have contended that in unilateral albuminuric retinitis only one kidney is diseased."

Aside from, or in addition to, the subjective and objective manifestations of the disease mentioned above, there occur those changes in the fundus described in the chapter relating to arteriosclerosis. Indeed, so closely are the two conditions associated—arteriosclerosis and nephritis—that it is extremely difficult to consider them separately, as it still remains a much mooted question in medical ranks whether arteriosclerosis causes nephritis or nephritis causes arteriosclerosis, so generally are the two found associated with one another. It has been conclusively proven that mechanical pressure can cause arteriosclerosis,¹ a fact which leads to the conclusion that the sclerosed artery is secondary to the nephritic disease causing a high arterial tension. On the other hand, certain investigators, Ophüle, for example, state emphatically that the ordinary red, granular contracted kidney is the *result* of general arteriosclerosis, and that in many of these cases the general arteriosclerosis is much more important in regard to the condition of the patient than the renal lesion which he may have at the same time. He says further that general arteriosclerotic lesions develop coexistently with arteriosclerotic lesions of the

1. See chapter on Arterial Pathology, page 38.

kidneys, but that there is no strict interdependence between the two processes. His conclusions are as follows:

"At the present time in many cases too much attention is paid to the kidneys clinically and anatomically. The so-called primary or genuine contracted kidney represents a disease of the kidney which is the result of arteriosclerosis in the terminal arterioles in this organ, is closely associated with general arteriosclerosis, and cannot be properly understood without due consideration of this fact."¹

However, while this question is one of importance in medical practice, it is of little concern under present consideration. The recognition of the existence of the disease is of far greater importance than a knowledge of its etiology, although it is quite true that the determination of the former may be dependent upon the latter.

A proper appreciation of this importance may have been instilled by the perusal of the foregoing part of this chapter and of the previous ones, but if it has not been, the following quotation from Parson's² "Diseases of the Eye" may serve the purpose:

"Degenerative Changes in the Retinal Vessels, apart from their interest as a local manifestation of disease, are of the utmost importance in general prognosis. They may be the first evidence of arteriosclerosis, pointing to the danger of cerebral hemorrhage, and indicating lines of treatment which may prolong life."

As an example, the following case may be cited:

Case 17899, Mrs. J. L. Housewife, age 53. Inclined to obesity. Has been coming to the writer for probably

1. Quoted from American Encyclopedia of Ophthalmology, page 1298.

2. Page 379.

ten years in regard to her refractive error which amounted to approximately one diopter of mixed astigmatism, with the usual presbyopia in one of her age. The visual acuteness, with proper lenses, has been until recently O. D. 15-20 partly, O.S. 15-13.

Her last call for an examination and change in lenses occurred about a year ago. Her complaint at that time was of minor character, and a change in lenses gave relief. She called again, however, about May 21, 1915, with the statement that vision in her left eye had suddenly decreased.

The ophthalmoscopic examination revealed an extensive destruction of tissue in the macular region of the left eye. No hemorrhage was visible, and other evidence led to the conclusion that the lesion was primarily choroidal, the eruption of tissue leading to destruction of the retinal membrane. Deposits of pigment were piled up.

The vision was reduced to 15-45 in the eye affected, a rather radical change from the 15-13 vision previously enjoyed. The other eye seemed approximately the same as before; if anything, slightly better vision was obtained in it.

The sphygmomanometer gave a pressure reading of about 220 mm. Hg. The patient was advised immediately to consult her family physician, which she did. He verified the writer's findings and sent her home to bed and quiet at once, and administered the necessary medicinal treatment and advice concerning diet.

His statement to the writer later contained the information that the patient was suffering from chronic interstitial nephritis and was just on the verge of a stroke of apo-

plexy. The patient called a week ago and it was found that the vision still remained about as before. There probably will be no *great* change in this respect, although her call today, July 1st, revealed 15-30 partly in the left eye.

Scattered on the fundus are several small hemorrhages about the size of a pinhead which have appeared since the previous examination. The blood-pressure has fallen to 170 mm. Hg.

While the prognosis in this case is now not particularly favorable, in all probability several years have been added to her life. Her age would encourage this conclusion, although Knies states that life is rarely prolonged more than one year, at the most two years, after the discovery of the retinal affection.

A similar ocular condition in one, say, twenty years younger, would justify a grave prognosis. The writer has in mind a man of about thirty, who was brought to him several years ago, by his father, for the purpose of seeing if there was any hope of recovering greatly destroyed vision. Bright's disease was known to be present.

The fundus was well covered with flame-shaped hemorrhages of a diameter equal to a disk-width, and numerous white patches. Vision was reduced to 15-30 O. U. The father ventured the information that the patient had suffered a "stroke" on the street, a month or two previous. Death occurred inside of six months.

Of course, these fundus changes were major ones and pointed unerringly to the presence of renal or vascular disease. Neither great acumen nor experience was required to recognize the gravity of conditions. The

minor changes are the ones more difficult of recognition and yet those in which the greatest good can be accomplished, for the patient can be warned of his condition and measures be taken to eliminate the disease at the point of orientation. These symptoms have been amply described in Chapter 6 and beautifully illustrated in the colored plate, but it may be well to reiterate a few of the more characteristic appearances that they may be strongly impressed upon the student's memory.

They consist, in the comparative order of their appearance, of loss of light streak along the veins near the point at which an artery crosses, and displacement of the vein in the direction of the arterial flow; indenture of the underlying vein by the stiffened artery passing over it; reduced caliber of a vein after being pressed upon in this manner by an artery; loss of light streak and marked tortuosity of the arteries; irregularity in the width and color of the arteries; the appearance of fine, white lines bordering the arterial or venous walls; minute aneurysms of the arteries; edema in the form of small whitish and grayish deposits upon the nerve-head and later upon the adjacent fundus; hyperemic retina and nerve-head; hemorrhages in the form of linear extravasations along the side of the vessels, and, finally, hemorrhages and exudates scattered over a wide area of the fundus and varying in size from minute spots to large red, flame-shaped or brilliant white areas, sometimes accompanied by pigment.

The final condition has been observed by the writer in a man of sixty in whom the fundus of one eye presented, in the ophthalmoscopic examination, very much the appearance of a sheet of blood, covering the entire

field. It is of interest to know that death occurred inside of six months.

It must be remembered, however, that these symptoms are not absolutely pathognomonic of albuminuria, that is to say, not an absolutely positive sign of nephritis, since a similar appearance has been found to be presented by intracranial tumor, anemia, syphilis, diabetes, lead encephalopathy and pachymeningitis.

Nevertheless, the frequency of this condition is indicated in the 104 cases presented by Dr. Luther P. Peter, in which ninety-four were due to chronic interstitial or chronic parenchymatous nephritis, nine of the remainder being due to syphilis and one to general arteriosclerosis.

Of the ninety-four mentioned, sixty showed neuro-retinitis, twenty-one simple retinitis, six hemorrhagic retinitis, three albuminuric retinitis, three papillitis, two neuro-retinitis combined with subconjunctival hemorrhage, one thrombosis of retinal vein and two negative. All of the nine cases of syphilis presented neuro-retinitis.

The frequency of retinal disease, according to different writers, ranges from seven to thirty per cent. Knies believed the lower figure nearer the truth, while Bulson¹ thinks that a much higher percentage than the sixteen or eighteen per cent mentioned by Edwards is more favorable. The writer's views accord with Bulson's.

These figures appear to be a very good indication of the relative frequency of the various ocular symptoms in kidney disease. As for the frequency of kidney disease in ocular symptoms, which is, of course, quite a different matter, Fox and Batroff² have reported one

1. J. A. M. Ass'n, Vol. LIX, page 1032.

2. Ophthalmic Record, October, 1908.

hundred cases of ocular hemorrhages in which fifty-three per cent were accompanied by nephritis in one form or another. Arteriosclerosis was also present in twenty-seven cases, but it can readily be seen that owing to the probability of nephritis, when resulting in high blood-pressure, causing arteriosclerosis, and to the hypothesis that renal arteriosclerosis may cause nephritis, the possibility of an accurate differential diagnosis by even a most skilled physician is rather remote. Following is the table submitted by Fox and Batroff:

Fig. 16

Table by Fox and Batroff

Case	Age	Ocular Diagnosis	Blood Pressure	H.	R.B.C.	Blood W.B.C.	Urine	General Diagnosis
1	58	Hemorrhagic Retinitis.	158 Mm.	70%	4,100,000	7,200	Albumin, Trace. No Casts.....	Arteriosclerosis.
2	35	Hemorrhagic Retinitis. Choked disc.....	240 Mm.	80%	3,960,000	7,000	Albumin and Casts.....	Chronic Parenchymatous Nephritis.
3	46	Anemia of Retina.....	152 Mm.	70%	3,200,000	6,800	Negative....	Secondary Anemia.
4	52	Hemorrhagic Retinitis. Subconjunctival Hemorrhage.....	178 Mm.	85%	4,040,000	7,000	Albumin. No Casts.....	Interstitial Nephritis
5	56	Retinal Hemorrhages..	240 Mm.	50%	3,740,000	7,800	Albumin and Casts.....	Interstitial Nephritis
6	34	Albuminuric Retinitis..	120 Mm.	62%	4,120,000	7,600	Albumin and Casts.....	Chronic Parenchymatous Nephritis.
7	36	Subconjunctival Hemorrhages.....	110 Mm.	73%	5,930,000	4,800	Negative....	Chloro-anemia.
8	52	Albuminuric Retinitis..	212 Mm.	78%	4,030,000	7,200	Albumin and Casts.....	Chronic Interstitial Nephritis.
9	38	Retinal Asthenopia and Hemorrhages.....	160 Mm.	68%	2,810,000	5,000	Negative....	Pernicious Anemia?
10	54	Arteriosclerosis of Retinal vessels.....	215 Mm.	78%	4,120,000	6,800	Albumin. Few Casts...	Arteriosclerosis.
11	36	Tumor of Orbit.....	105 Mm.	85%	5,960,000	6,400	Negative....	Orbital Tumor.
12	58	Hemorrhagic Retinitis..	136 Mm.	90%	4,390,000	5,600	Negative....	Arteriosclerosis.
13	35	Hemorrhagic Retinitis..	110 Mm.	76%	3,840,000	5,800	Negative....	Anemia and Intestinal Autotoxemia.
14	53	Subconjunctival Hemorrhages.....	230 Mm.	90%	6,050,000	5,000	Albumin. Few Casts...	Interstitial Nephritis
15	48	Diabetic Hemorrhagic Retinitis.....	120 Mm.	90%	5,350,000	7,200	Glucose....	Diabetes,

BLOOD PRESSURE IN OCULAR WORK

Case	Age	Ocular Diagnosis	Blood Pressure	H.	R.B.C.	Blood W.B.C.	Urine	General Diagnosis
1638		Edema of Retina.....	110 Mm.	95%	5,050,000	7,400	Negative....	Intestinal Autoin- toxication.
1745		Subconjunctival Hemor- rhage.....	160 Mm.	84%	4,120,000	7,600	Albumin and Casts.....	Early Interstitial Nephritis.
1857		Albuminuric Retinitis. Hemorrhages.....	155 Mm.	92%	5,060,000	6,500	Albumin and Casts.....	Advanced Inter- stitial Nephritis.
1929		Retinal Hemorrhages. Double Choked Disc...	135 Mm.	85%	4,020,000	8,000	Negative....	Syphilis.
2058		Retinal Hemorrhages..	142 Mm.	80%	3,910,000	7,400	Albumin. Negative. Hyalin Casts.	Interstitial Nephritis
2178		Hemorrhage of Right Macula.....	170 Mm.	75%	4,800,000	7,200	Albumin. No Casts....	Senile Arterioscle- rosis.
2246		Anemia of Retina. Old Hem.....	152 Mm.	67%	3,650,000	6,000	Albumin. No Casts....	Symptomatic Anemia.
2349		Albuminuric Retinitis..	135 Mm.	85%	4,420,000	7,200	No Albumin. Casts.....	Interstitial Nephritis
2454		Hemorrhagic Retinitis..	236 Mm.	80%	4,200,000	7,400	Albumin. Casts.....	Interstitial Nephritis
2543		Albuminuric Retinitis..	190 Mm.	83%	4,620,000	8,000	Albumin and Casts.....	Secondary Contract- ed Kidney.
2636		Subconjunctival Hemor- rhage.....	135 Mm.	90%	4,700,000	7,800	No Albumin. Casts.....	Kindey of Pregnancy
2732		Albuminuric and Hem- orrhagic Retinitis.....	193 Mm.	85%	5,010,000	7,200	Albumin and Casts.....	Chronic Parenchy- matous Nephritis.
2830		Hemorrhagic Retinitis..	125 Mm.	75%	4,570,000	8,000	Negative....	Secondary Anemia. Overwork and Ex- haustion.
2948		Hemorrhagic Retinitis..	166 Mm.	72%	4,730,000	9,000	Albumin. Casts.....	Interstitial Nephritis
3052		Albuminuric Retinitis..	235 Mm.	60%	3,400,000	6,600	Albumin and Casts.....	Late State Interstitial Nephritis.
3134		Anemia of Retina with Small Hemorrhages....	130 Mm.	65%	2,510,000	130,000	Albumin. No Casts....	Leukemia. D
3264		Hemorrhagic Retinitis..	204 Mm.	60%	4,350,000	7,400	Albumin and Casts.....	Chronic Interstitial Nephritis.
3345		Hemorrhagic Retinitis..	220 Mm.	82%	4,110,000	7,600	Albumin and Casts.....	Chronic Interstitial Nephritis.
3436		Ocular Headache. Sub- conjunctival Hemor- rhage.....	122 Mm.	83%	4,180,000	7,400	Oxaluria....	Lithemia.
3564		Retinal Hemorrhage...	166 Mm.	92%	5,001,000	7,600	Albumin. No Casts.	Arteriosclerosis.
3654		Subconjunctival Hem- orrhage.....	178 Mm.	96%	4,992,000	8,200	Albumin. No Casts....	Arteriosclerosis.
3737		Subconjunctival Hem- orrhage.....	142 Mm.	78%	5,001,000	7,600	No Albumin or Casts. Uric Acid....	Lithemia.
3847		Subconjunctival Hem- orrhage.....	170 Mm.	90%	4,060,000	7,400	Albumin and Casts.....	Arteriosclerosis.
3944		Subconjunctival Hem- orrhage.....	160 Mm.	85%	4,120,000	8,000	Albumin and Casts.....	Chronic Parenchy- matous Nephritis.

Case	Age	Ocular Diagnosis	Blood Pressure	H.	R.B.C.	Blood W.B.C.	Urine	General Diagnosis
40	42	Hemorrhagic Retinitis..	152 Mm.	88%	4,200,000	7,800	Albumin. No Casts....	Retinal and General Arteriosclerosis.
41	58	Albuminuric Retinitis..	158 Mm.	70%	3,980,000	8,000	Albumin and Casts.....	Arteriosclerosis.
42	59	Subconjunctival Hem- orrhage.....	149 Mm.	84%	4,010,000	6,500	Albumin and Casts.....	Chronic Interstitial Nephritis.
43	62	Sclerosis of Retinal Vessels.....	162 Mm.	95%	4,832,000	6,800	Albumin. No Casts....	Arteriosclerosis and Presenility.
44	29	Albuminuric Retinitis..	148 Mm.	82%	4,100,000	7,600	Albumin and Casts.....	Chronic Parenchy- matous Nephritis.
45	72	Unilateral Retinal Hem- orrhages.....	232 Mm.	80%	3,720,000	7,200	Albumin and Casts.....	Arteriosclerosis and Arteriosclerotic Kidney.
46	36	Colloid Degeneration of Retinal Vessels.....	132 Mm.	68%	3,840,000	4,800	Albumin, Trace. Casts Few.	Beginning Retinal Degeneration.
47	45	Retinal Hemorrhage...	144 Mm.	82%	4,120,000	6,800	Albumin. No Casts...	Early Arterioscle- rosis.
48	43	Macular Hemorrhage..	136 Mm.	88%	4,020,000	7,200	Albumin. No Casts....	Early Cirrhotic Kidney.
49	54	Retinal Hemorrhage...	255 Mm.	55%	3,640,000	6,800	Albumin and Casts.....	Chronic Interstitial Nephritis.
50	63	Silver Wire, Retinal Vessels.....	140 Mm.	75%	4,114,000	6,200	Albumin. No Casts....	Arteriosclerosis.
51	56	Retinal Congestion, slight Hem. and Edema	148 Mm.	92%	5,001,000	7,400	No Albumin. No Casts....	Passive Cerebral Congestion Result of Aneurysm.
52	36	Subconjunctival Hem- orrhage.....	144 Mm.	94%	5,020,000	7,800	No Albumin. No Casts. Uric Acid.	Lithemic Hyperten- sion Progressing to- ward Cirrhotic Kidney.
53	33	Albuminuric Retinitis..	122 Mm.	60%	3,812,000	6,800	Albumin and many Casts..	Chronic Parenchy- matous Nephritis.
54	53	Albuminuric Retinitis..	264 Mm.	76%	4,020,000	7,200	Trace Alb. Few Casts...	Chronic Interstitial Nephritis.
55	46	Diabetic Hemorrhagic. Retinitis.....	110 Mm.	84%	4,760,000	7,400	Glucose Pres- ent. Albumin and Casts Negative.	Diabetes.
56	44	Hemorrhagic Retinitis..	185 Mm.	90%	4,328,000	7,200	Albumin Neg- ative. Casts Negative....	Sudden Vascular Rupture under ex- citement in case of Early Atheroma.
57	66	Cataract and Conj. Hemorrhage.....	164 Mm.	66%	3,600,000	5,400	Albumin and Casts Present	Chronic Interstitial Nephritis.
58	43	Retinal Hemorrhage...	232 Mm.	95%	5,100,000	7,800	Albumin. Casts Present	Chronic Interstitial Nephritis.
59	46	Retinal Hemorrhage...	260 Mm.	75%	5,340,000	11,000	Albumin and Casts.....	Chronic Interstitial Nephritis. Reduced Pressure to 180.
60	27	Recurrent Iritis and Retinal Hemorrhage...	126 Mm.	90%	4,800,000	7,200	Alb. Trace. Casts Neg...	Specific Iritis.
7	40	Retinal Hemorrhage...	188 Mm.	88%	4,621,000	7,800	Alb. Neg. Casts Neg...	Arteriosclerosis.

Case	Age	Ocular Diagnosis	Blood Pressure	H.	R.B.C.	Blood W.B.C.	Urine	General Diagnosis
62	58	Retinal Hemorrhage...	178 Mm.	90%	4,392,000	8,000	Alb. Trace. No Casts....	Arteriosclerosis.
63	50	Retinal Hemorrhage...	148 Mm.	87%	4,862,000	7,600	Alb. Trace. No Casts....	Interstitial Nephritis
64	63	Glaucoma. Subcon- junctival Hemorrhages.	225 Mm.	96%	5,010,000	8,200	Alb. Trace. Few Casts...	Glaucoma and Inter- stitial Nephritis.
65	59	Retinal Hemorrhages...	265 Mm.	90%	4,110,000	7,600	Albumin and Casts.	Interstitial Nephritis
66	69	Retinal Hemorrhages...	195 Mm.	69%	3,800,000	7,200	Alb. Excess. Casts numer- ous.	Moderate Cardiac Dilatation. Chronic Nephritis.
67	38	Retinal Hemorrhage...	160 Mm.	76%	4,160,000	7,600	Alb. Trace. Few Casts...	Chronic Nephritis.
68	45	Retinal Hemorrhage...	195 Mm.	94%	4,980,000	7,800	Alb. Trace. No Casts....	Arteriosclerosis.
69	54	Retinal Hemorrhage...	212 Mm.	91%	4,001,000	7,600	Alb. Trace. Casts Few...	Chronic Interstitial.
70	54	Retinal Hemorrhage...	226 Mm.	95%	4,872,000	7,200	Alb. Trace. Few Hyalin..	Chronic Interstitial.
71	58	Hemorrhagic Retinitis.	164 Mm.	72%	4,211,000	8,000	Alb. Trace. Few Casts...	Arteriosclerosis.
72	36	Hemorrhagic Retinitis. Choked Disc.....	242 Mm.	83%	4,480,000	7,600	Much Alb. Many Casts.	Chronic Parenchy- matous Nephritis.
73	53	Retinal and Subcon- junctival Hemorrhage...	179 Mm.	86%	4,040,000	7,400	Alb. Trace. Few Casts...	Interstitial Nephri- tis.
74	57	Hemorrhagic Retinitis.	243 Mm.	54%	3,622,000	6,200	Albumin and Casts.	Interstitial Nephri- tis.
75	36	Albuminuric and Hem- orrhagic Retinitis....	144 Mm.	62%	3,921,000	6,800	Albumin and Casts.	Chronic Parenchy- matous Nephritis.
76	54	Arteriosclerosis of Reti- nal Vessels. Small Hemor.....	215 Mm.	78%	4,013,000	7,400	Alb Trace. Few Casts...	Interstitial Nephritis
77	60	Hemorrhagic Retinitis.	130 Mm.	92%	4,382,000	5,600	Alb. Trace. No Casts	Arteriosclerosis.
78	52	Subconjunctival Hem- orrhages.....	234 Mm.	84%	5,020,000	5,800	Albumin. Few Casts...	Interstitial Nephritis
79	46	Subconjunctival Hem- orrhages.....	164 Mm.	85%	4,320,000	7,600	Alb. Trace. Occasional Cast.	Early Interstitial Nephritis.
80	79	Macular Hemorrhage..	178 Mm.	78%	3,900,000	6,200	No Albumin Few Casts Albumin....	Senility. Arterio- sclerosis.
81	54	Retinal Hemorrhages..	235 Mm.	81%	4,196,000	8,000	Albumin Trc. Many Casts..	Chronic Interstitial Nephritis.
82	31	Albuminuric and Hem- orrhagic Retinitis....	194 Mm.	86%	4,680,000	7,200	Much Alb. Many Casts..	Chronic Parenchy- matous Nephritis.
83	65	Hemorrhagic Retinitis.	232 Mm.	62%	3,420,000	6,600	Albumin. Many Casts..	Advanced Intersti- tial Nephritis
84	64	Hemorrhagic Retinitis..	168 Mm.	86%	4,021,000	6,800	Albumin. No Casts....	Arteriosclerosis.

Case	Age	Ocular Diagnosis	Blood Pressure	H.	R.B.C.	Blood W.B.C.	Urine	General Diagnosis
85	38	Subconjunctival Hemorrhage.....	144 Mm.	78%	5,010,000	7,200	No Albumin or Casts. Uric Acid...	Lithemia.
86	43	Retinal Hemorrhage...	153 Mm.	87%	4,121,000	8,000	Alb. Trace. No Casts....	Early Arteriosclerosis.
87	71	Retinal Hemorrhage...	230 Mm.	81%	3,890,000	4,800	Alb. Trace. Few Casts...	Arteriosclerosis and Arteriosclerotic Kidney.
88	55	Retinal Hemorrhage...	255 Mm.	61%	3,640,000	6,800	Albumin and Casts.....	Chronic Interstitial Nephritis.
89	57	Retinal Hemorrhage...	184 Mm.	64%	3,890,000	7,200	Albumin and Casts.....	Chronic Interstitial Nephritis.
90	28	Albuminuric Retinitis...	146 Mm.	73%	4,001,000	7,600	Much Alb. Many Casts.	Chronic Parenchymatous Nephritis.
91	57	Retinal Hemorrhage...	156 Mm.	96%	5,010,000	8,000	Alb. Faint Trace. No Casts.....	Arteriosclerosis.
92	33	Albuminuric and Hemorrhagic Retinitis.....	124 Mm.	72%	3,920,000	7,800	Much Alb. Many Casts.	Chronic Parenchymatous Nephritis.
93	44	Hemorrhagic Retinitis...	184 Mm.	88%	4,231,000	7,200	Alb. Negative Casts Few...	Early Arteriosclerosis.
94	67	Subconjunctival Hemorrhage.....	165 Mm.	68%	3,920,000	7,400	Alb. Present. Few Casts...	Chronic Interstitial Nephritis.
95	43	Retinal Hemorrhage...	228 Mm.	84%	4,200,000	7,600	Albumin and Casts Present	Chronic Interstitial Nephritis.
96	47	Retinal Hemorrhage...	196 Mm.	89%	4,021,000	8,000	Alb. Trace. Casts Numerous.....	Early Arteriosclerosis. Cirrhotic Kidney.
97	54	Retinal Hemorrhage...	218 Mm.	92%	4,211,000	6,800	Alb. Trace. Casts Few...	Chronic Interstitial Nephritis.
98	50	Retinal and Subconjunctival Hemorrhage...	152 Mm.	90%	4,078,000	9,000	Alb. Trace. No Casts....	Generalized Arteriosclerosis.
99	31	Albuminuric and Hemorrhagic Retinitis.....	148 Mm.	66%	4,112,000	8,200	Alb Excess. Num. Casts...	Chronic Parenchymatous Nephritis.
100	63	Retinal Hemorrhage...	22 Mm.	97%	4,820,000	76,800	Alb. Trace. Casts Numerous.....	Chronic Interstitial Nephritis.

As it has been pointed out that the fundus changes are not always positive indications of kidney disease, so should it be emphasized that the absence of albumin from the urine does not entirely exclude the disease. Knies and others insist that albuminuria should be excluded only after repeated examinations, and so a physician's negative report upon a case sent to him should be accepted only after a series of examinations have been made.

Concerning muscle paralyses due to albuminuria, it has been the writer's experience that the external rectus muscle is the more frequently affected, but it must be admitted so few cases undeniably due to albuminuria have been observed by him that he is not in a position to state positively whether other muscles of the orbit can be similarly affected, and if so, what the relative frequency may be.¹ Most of the cases of the so-called spontaneous imbalance observed by him have been due to syphilis.

When due to albuminuria, the lesion develops rapidly and sometimes to an exaggerated degree. It decreases and disappears also with comparative rapidity. Knies' observation concerning this subject is as follows²:

"Little is found in literature concerning paralyses of the ocular muscles in albuminuria. Finlayson (*Jahr. f. Aug.* 1877, p. 240) reports right abducens paralysis due to a hemorrhage into the pons. Foerster (*l.c.*) makes no mention of these paralyses. They are so frequent, however, that in every case of sudden or rapidly developing paralysis of the ocular muscles with the character of basilar, root, or nuclear paralysis, the urine should be examined for albumin. The cause generally appears to consist of a hemorrhage in the region of the nerve roots or nuclei, possibly even in the nerve itself. Sclerosis also occurs in the nerves of the ocular muscles, as Leber demonstrated anatomically in the abducens.

"The paralyses usually recover rapidly with or without treatment, but often undergo relapses in the same or other muscles. I have recently seen three cases of this kind in rapid succession: 1. Abducens paralysis as the sole eye symptom in albuminuria of fifteen years' stand-

1. Hansel and Rebner, in their excellent work, "Ocular Muscles," deal generally with this subject on pages 43 and 62, and all their conclusions appear to be that, considering ocular palsies from all causes, the external rectus leads in numerical order, the reason advanced being that its long course from the base of the brain renders it peculiarly liable to arrest of function during disease. The reader is referred to that work.

2. Page 307.

ing, which had been left after typhoid fever; it relapsed twice in a few months, and then the patient died. 2. A left trochlear paralysis—on the right side hemorrhages were found in the optic nerve—in contracted kidneys of unknown duration; death in six months. 3. A complicated external ophthalmoplegia in a man of twenty-four years; the albuminuria succeeded typhoid fever two years before. On the right side the trochlearis was first attacked; then, while this was recovering, the internal rectus and the other external muscles supplied by the motor oculi; finally, attacks of unilateral and bilateral ptosis. The paralyzes recovered rapidly; the right internus remained paretic longest, alternating with the inferior rectus. Soon afterward right ptosis again developed for several days, and then I lost sight of the patient. The ophthalmoscopic appearances and the internal ocular muscles were always entirely normal, and syphilis could be excluded with certainty.

“The muscular paralyzes also appear to be terminal symptoms of albuminuria, and are certainly indicative of changes in the cerebral vessels similar to those which are found in the retina.”

As has elsewhere been pointed out by the writer, these muscle disturbances may arise from several secondary causes. When strictly of a paralytic nature, they may be due to destruction of nuclear cells, which results in the non-transmission to the nerve of impulses sent out from the brain; they may be due to an actual destruction of the fibers of the nerve itself; pressure on the nerve, resulting from an accumulation of an exudate or blood-clot in the nuclear region or along the course of the nerve, notably in or beside the cavernous sinus where the arrangement of the third, fourth and sixth nerves as they accompany the

internal carotid artery renders them peculiarly susceptible to external influences (Fig. 4); aneurysms of the internal carotid or adjacent arteries as mentioned on page 91.

It is conceivable, and may have been demonstrated by others, that the muscle disturbances are not necessarily paralytic. They may be spasmodic, being due to an irritation of the nerve fibers at any point from their nuclear, or even cortical inception, to their muscular insertion. A pathologic condition of adjacent tissues, resulting in the liberation of toxic products, can bring this about, particularly if morbid degeneration of the nerve itself or its nuclear area has set in.

This may explain the occasional transition of an internal strabismus into an external one, or *vice versa*. In the primary stage of the disease, the nerve is irritably stimulated and a spasm results. As the area of the destroyed and non-active tissue increases, the nerve gradually loses its functioning power, and total incapacity eventually sets in. The muscle naturally declines into a non-active state and the eye rotates to the opposite side.

In finality, there should be pointed out a subjective symptom of albuminuria and analogous conditions of which no specific mention has been made in the preceding pages, altho the writer has suggested it elsewhere.¹ This consists of a peculiar sense of blur, experienced by the patient, which lenses fail to obviate.

It can easily be demonstrated in the average healthy case that an eye artificially myopic half a diopter, that is, an eye which has before it a plus .50 lens in addition to lenses which correct all refractive errors, should see 15-20

1. Keystone, April 8, 1915: The Value of the Blood-Pressure Test to the Optometrist. Case No. 17918. Keystone 1-14-15: Rapid Changes in Ocular Refraction.

if its vision without such a lens is 15-13, or even 15-15.¹ As a rule, if the eye can see 15-15, or better, with such a glass, it indicates that there is an under-correction of hyperopia or an over-correction of myopia, but this is not true in the class of cases in mind.² In these a certain defect may be demonstrated skiametrically, and sustained in the subjective test by a rejection by the patient of increased plus or decreased minus lenses, and yet normal vision (in so far as the actual deciphering of the letters is concerned) maintained, even tho a plus .50 or even a plus .75 (rarely a plus 1.00) be added to the correction previously determined upon.

The criterion in these cases, such as it is, consists of the patient's sensation *upon the removal of the plus .50 lens after sufficient plus has been added to effect a blur resulting in 15-20 vision. If, after vision has been thus cut down to 15-20, the removal of a plus .50 still leaves a blur which the incorporation of a minus .25 lens, or its equivalent,³ with the remaining correction in the trial frame does not totally eliminate, it may safely be assumed that the blur is pathological and not optical.*

To illustrate, let there be supposed a case in which the skiascope has revealed 1.00 D. of hyperopia. Let it be further assumed that the patient is a healthy individual in all respects and that the subjective test sustains the skiascopic findings—that is, there is actually present just 1.00 D. of hyperopia. If the plus 1.00 D. lens gives 15-13 vision and that vision is perfectly clear, the addition of a plus .50 lens will prevent the reading of any letters smaller than the 15-20 line. The addition of plus .25 more

1. It should be explained that the distance of the writer's test chart is fifteen feet, and that the average eye can see the 15-13 line when properly corrected. Of course, amblyopic eyes which cannot see 15-15, or better, either with or without glasses, are excluded from this test, as a rule. As the degree of amblyopia increases it becomes increasingly difficult to make the test.

2. It must be accepted that the means at the writer's command exclude the probability of an overcorrection of myopia or an undercorrection of hyperopia in such cases. A number of tests, including the fogging system, are utilized to exclude the probability of spasms, etc.

3. Naturally, a reduction of the plus lenses.

will exclude even this line, resulting in 15-30 vision. The removal of the plus .50—and, of course, of the plus .25 if it still remains—will result in a return to perfectly clear vision.

On the other hand, suppose the health of the patient is in doubt. With a plus 1.00 before the eye, vision is fairly clear, and deciphering of the 15-13 line easily accomplished, altho the patient does not exclaim that "everything is perfectly clear and sharp." A plus .50 is now added to the plus 1.00, and the patient asked concerning the result. If the case is of the type in mind, the answer is exasperatingly inconsistent. Yes, *the last line can still be read*, but everything is foggy, misty. The addition of a plus .25 to the above "does not seem to change it much, tho it does not seem to be quite as good." Fifteen-twentieths or even 15-15 vision may still be maintained! (Note that in the healthy case the addition of the plus .25 blurs to 15-30.)

*If, upon the removal of the plus .50, the patient still senses a blur and states that "things don't seem quite as clear as they should be," and this "blur" does not totally disappear upon the removal of the plus .25, there can scarcely remain a doubt that there are present abnormal conditions other than refractive.*¹ This existence of 15-20, or better, vision when a plus .75 lens has been added is proof positive that the eye is not over-corrected, and the presence of a blur after it has been removed just as conclusively demonstrates the presence of an abnormality not optical.

To give, in such a case, lenses just short of those which would prevent 15-15 or 15-13 vision² is fatal to the

1. Of course, it is understood that all astigmatism must be accurately corrected in this test. Otherwise, no spherical lens will give very clear vision.

2. It will be noted that in the illustration this lens would be plus 1.25 at least, perhaps plus 1.50.

comfort and satisfaction of the patient, for they will almost invariably be too strong if plus, and too weak if minus, and will tend to make a bad matter worse. Yet that is exactly what one is tempted to do unless there are other symptoms discovered which discourage such action. It is in just such cases that the patient finally becomes disgusted with the result and tries other refractionists, or exhausts the patience of the first one by insisting upon the frequent changing of lenses.

The local cause of this blurring is not positively known to the writer, but it has been found to be associated with albuminuria in many cases. It has also been discovered in other diseases affecting the retina of the eye, notably syphilis, but the condition seems to be most pronounced in nephritis and lends itself readily to an association with the symptomatology of this class of diseases.

When the patient relates the effect as it appears to him, stating that he "sees everything, but somehow things don't seem just right; they seem cloudy, hazy, just as tho one is looking through smoke," one is inclined to believe that the local cause consists of the presence of an obstructing material in the vitreous, but when a careful ophthalmoscopic examination reveals media as clear as the normal, this theory is weakened.

Two remaining hypotheses are reasonable; the one, that the condition is due to a dulling of the nerve elements, either peripheral, nuclear or central, caused by the toxic products liberated by the diseased organ (the kidney); and the other, that the mechanical pressure of the ocular contents upon the retina produces a sort of "pressure anesthesia." This latter theory appears to be more or less substantiated by the commonly accepted fact that the intraocular pressure parallels the general arterial

pressure, and since it is the rule for the arterial pressure to be elevated in kidney disease, it naturally follows that there is also a concomitant rise in the intraocular pressure.

In all probability, all these conditions lend themselves as causes of the blurred condition, and when the latter symptom is accompanied by a rise in the blood-pressure, the conclusion that there are present abnormal conditions other than refractive is founded upon very substantial premises.

Prognosis in these cases is not particularly encouraging, either in regard to the ocular or the general welfare, as the blur so frequently presages retinal changes of moment, or an early noticeable decline in health. Upon detection of the symptom, especially if it is coexistent with fundus changes and an abnormal blood-pressure reading, the patient should immediately be turned over to the physician for further examination.

The result of glasses in such cases is problematical in so far as relief from the symptoms complained of—blurred vision, headache, indigestion, vertigo, usually—is concerned, but most emphatically they should be given if the slightest correctable defect exists, the purpose being to remove all strain possible and thereby reduce the possibility of ocular hemorrhage.

CONDITION OF BLOOD-PRESSURE. An elevated arterial tension so frequently exists in albuminuria that it may be considered almost pathognomonic of the disease. Hirschfelder¹ says, "High blood-pressure is common to both parenchymatous and interstitial cases," and reference to the table by Fox and Batroff will show that forty-seven out of fifty-seven cases of kidney diseases possessed high tension. In the table presented by Dr. Luther

1. Diseases of the Heart and Aorta, pages 39, 40.

Peter, eighty-four of the ninety-four cases mentioned as nephritic gave a high pressure reading.

In the nephritic cases submitted by Fox and Batroff the highest tension was 265 mm. and the lowest 120 mm. In that by Peter, the highest was 260 mm. and the lowest 122 mm. In the former table, tension under 140 mm. occurs only six times, and in the latter only twice.

Without seeking further, it is clearly evident, from these cases, that hypertension is the rule in nephritic disease. It is very probable, too, that the majority of the cases of hypertension encountered in optometric practice are nephritic. The material submitted does not necessarily prove this supposition to be a fact, but the evidence certainly sustains it as a reasonable contention.

It is appropriate, and advisable, to close this chapter with a word of caution. A high pressure reading, obtained at a single sitting, especially the first time a patient calls, should not be accepted as a permanent condition. It must be substantiated one or more times, preferably the latter. Dr. Peter has said, "The same patient will show marked variations in blood-pressure at different times. He will come to the office one day with a pressure of 200 to 225 mm., and another day with 150 mm." While the writer has never encountered such radical differences, it is not impossible that they may occur. The case must be studied and a general average taken, rather than a single reading.

Uremia, a form of nephritis, characterized by the accumulation in the blood of urea, the chief solid constituent of urine, affects the eye apparently with much less frequency than albuminuria.

Temporary amblyopia or amaurosis seems to be the principal ocular effect, muscular effects occurring less

often; according to Knies, about one per cent. This author states that the disturbance is usually bilateral, develops rapidly and generally results in total blindness. The restoration of sight usually takes twenty-four to thirty-six hours.

The affection is commonly preceded by headache; during the attack the ophthalmoscopic evidence seems to be negative. The reaction of the pupil to light is retained.

It is seldom accompanied by retinal disease unless the latter exercises priority. In this connection, it may be well to state that whereas retinal affections usually accompany *chronic* renal disease, uremic disorders of sight more frequently accompany *acute* nephritis.

The ocular symptoms other than amblyopic or amaurotic consist of mydriasis or myosis (these usually when accompanied by other diseases), pallor of the fundus (due to vascular spasms), convulsions in which the ocular muscles may be involved (convergence, etc.), optic neuritis (apparently rare), hemeralopia, etc.

CONDITION OF BLOOD-PRESSURE. This symptom is generally stated to run parallel with the severity of the disease. This holds good only until a collapse is imminent. Hirschfelder states, "In uremia, blood-pressure rises at the beginning of the attack, but may gradually fall a few days before a fatal termination. Gradual fall in blood-pressure also accompanies amelioration."

The consensus of opinion seems to be that the blood-pressure may rise to 300 mm.

CHAPTER 9.

DISEASES OF THE GENERAL SYSTEM

That the eye should take part in the general malaise of constitutional diseases is to be expected. Both the excessive use to which the organ is put and the delicacy of its tissues predispose the organ to great susceptibility. The extreme sensitiveness of its neural connections encourages surprise that its participation is not more frequent and more pronounced.

Perhaps the association of the ocular and the general symptoms is even more prevalent than the writer supposes. The general practicing physician is in a better position to answer this question than the optometrist, since he, naturally, not only has more of such cases in his practice, but he is able to recognize a much higher percentage of them. It appears customary, and natural, that, upon diagnosis of the major disease, local ocular symptoms are assumed by him to be dependent upon the latter, and when they disappear upon treatment of the underlying cause, it is taken as a matter of course. Such cases the optometrist comparatively seldom has the opportunity to follow or even to hear of, and for this reason it can readily be imagined that the association of ocular symptoms with the diseases in mind is more frequent than the writer's experience would indicate, or than ophthalmological literature intimates, since the

same reasoning applies to the ophthalmologist as to the optometrist, only to a lesser degree.

However this may be, there is an association and it is proper to consider it.

Anemia exercises great influence in optometric work, owing both to its effect upon the eye directly and to the close similarity between its symptoms and those of eye-strain.

Neglecting for the moment the functional disturbances due to the condition, the ocular lesions which may be attributed to it vary from fundus changes invisible macroscopically to the profuse hemorrhagic appearance sometimes attending pernicious anemia. Knies states that it is only in the advanced cases that the papilla is notably paler, but says that it may even be chalky white, the blood stream lighter, and pulsation of the vessels occasionally visible, while the same author mentions that "a rather striking fact is the great frequency of congestion of the conjunctiva, also called dry catarrh, in anemia of all kinds. One of its main causes is probably insufficient sleep or insomnia."

Of the more spectacular evidences he gives neuritis, retrobulbar neuritis and atrophy of the optic nerves, and states that the ophthalmoscopic appearances may resemble those of albuminuria, even tho there may be no evidence of albumin in the urine, altho on another page he suggests that these latter changes are more or less accidental complications.

Wood,¹ also, states that "Deposits in the retina and choroid, and even neuro-retinitis with hemorrhages, are also seen now and then. The specific new formations in

1. American Encyclopedia of Ophthalmology, Vol. I, page 420.

the lymph glands and spleen, particularly those that mark the so-called leukemia, furnish another example of intoxication, with definite ocular changes. A bilateral retinitis, with numeral hemorrhages (resembling the albuminuric form), is the most important of these, due to fatty and other alterations in and about the minute retinal capillaries. Fundus and vitreous changes have also been discovered in the advanced stages of the disease."

Turning now to the local functional and subjective evidences of the condition, it appears commonly agreed that they consist chiefly of restricted accommodative ability, pain and exhaustion upon close application of the eye, retinal asthenopia, hemeralopia, narrowing of field of vision and paroxysmal loss of sight (the temporary "blind," "black" spells so often mentioned by the patients, probably due to recession of blood from the eye, caused by its accumulation in other parts of the body, or, perhaps, by a spasm of the vessel walls).¹

The general symptoms are:² "pallor of the skin and mucous membrane, weakness and lassitude, shortness of breath, palpitation, a tendency to fainting, and usually also gastro-intestinal disturbances, headache and neuralgia."

It is clear to all experienced refractionists how closely similar many of these symptoms are to the symptoms of eye-strain. In a high percentage of cases an accurate differential diagnosis, based solely upon a recitation of the symptoms by the patient, is little short of impossible, and it becomes absolutely essential to introduce objective methods in order to ascertain the true facts in a given

1. Since it has been stated that the vessels upon the fundus contain no muscle fibers, the spasm must take place before their entrance into the eye.

2. Gulland, Pathology of the Blood, *Encyclopædia Britannica*, Vol. IV, page 83.

case.¹ Of course, the ophthalmoscopic examination may yield information, but oftentimes its revelations are negative.

The writer above quoted (Gulland) says, "The diagnosis depends ultimately in all cases upon the examination of the blood." For obvious reasons, at the present time an actual blood-count is impracticable in optometric practice, but the ascertainment of the *blood-pressure* is entirely practicable, and this method may be employed. The value of this test was clearly demonstrated in Case No. 17995, cited in the series presented in the *Keystone Magazine of Optometry* in the issue of May 6, 1915. In this instance, the patient was evidently suffering from temporary secondary anemia due to the sanguineous operation, but the immediate mechanical evidence was probably precisely the same as tho it was due to other causes.

CONDITION OF BLOOD-PRESSURE. Hypotension exists in anemia, being due to either the reduction in amount of blood or of its hemoglobin constituent, or to both. Perhaps, when the number of red corpuscles is reduced and the toxic-carrying properties of the fluid thereby curtailed, the accumulation in the system of the poisonous products of metabolism tends so to affect the vasoconstrictor nerves, or their centers, that the lumen of the vessels is widened and the pressure thereby reduced. The lessened speed of the blood stream in consequence of this fact would complete the vicious circle and perhaps explain, in part, the difficulty of remedying the disease known as *pernicious anemia*.

1. Again the writer feels constrained to point out that such a diagnosis is not to be offered the patient except in a general way. However, it is highly desirable that some sort of logical conclusion be arrived at in the operator's mind concerning the true state of the patient's condition, that efforts to relieve the symptoms may be intelligently directed, for precisely the same reason that it is desirable to know whether or not, in a given case, blurred vision is due to faulty refraction or to destroyed functioning power of the neural system. But until such a time as the optometrist is in a position to *know*, it is rather the physician's province to inform the patient of his or her condition,

However this may be, it is certain that the curtailment of the integrity of the blood, whether involving its chemical or its mechanical properties, may produce many of the symptoms of anemia previously enumerated. Their severity will depend, of course, upon the extent of the disease and upon *other aggravating factors*.

The acknowledgment that eye-strain may be one of these illuminates many of the remarkable cases of relief accomplished in refractive work. It is clear that in these cases the existing eye-strain constitutes the "last straw," and that its removal is sufficient to sway the scales in favor of relief. This occurs in those delicately balanced individuals who are always on the verge of collapse in one form or another. It especially applies to those afflicted with that form of anemia known as *chlorosis*. This is present in only the female sex and appears chiefly between the ages of fifteen and twenty-five. It is principally in such cases that correcting lenses are found to accord a relief markedly disproportionate to the defect discovered, and it has been the experience gained in these that has led the writer to conclude that the lowest pressure compatible with good health and comfort (all other things being equal, of course) should be 110 mm. instead of 100 mm., as usually stated in text-books.

In other words, pressure of 110 mm. or less should be viewed with suspicion. If a patient with such a pressure complains of headache, pain and discomfort upon use of the eyes, and a moderate refractive or muscular defect is discovered, the chances are that lenses will give pronounced relief if otherwise the patient is apparently in good health. If *not* in good health, or the pressure is lower than 110 mm., prognosis should not be unduly optimistic. In all probabilities, only partial relief will be given by lenses. This is true sometimes even when

the pressure is 110 mm. or a little over, if such a pressure is inconsistent with the age, sex, physique, etc., of the patient. For example, 110 mm., while perhaps normal in a healthy woman of twenty or thirty years, might be abnormal in one of fifty or sixty years.

It may be stated upon good authority that pressures under 100 mm. are always considered pathological, except in children or very youthful individuals.

It is apparent, then, that sphygmomanometric readings between 100 mm. and 110 mm. constitute a section of the manometer scale of vital importance in refractive work. It is here that the greatest successes occur, but it is also an area containing many indifferent results, or even keen disappointments.

It appears that in *pernicious* anemia, arterial tension may be as low as 90 mm.; perhaps lower.

Diabetes Mellitus—**GLYCOSURIA**—while a factor comparatively seldom entering into optometric practice, sometimes intrudes with disconcerting effect.

A disease characterized by a superabundance of sugar in the urine, and one involving practically the entire system, often terminating in death, it is to be expected that its effect upon the delicate ocular tissue may, with relative frequency, be pronounced.

These effects, as given by Wood, Knies and others, consist, objectively, of pareses of the ocular muscles, iritis, cataract, failure of accommodation, refractive changes, retinitis (simple, hemorrhagic and punctate), optic neuritis, vitreous opacities, detachment of the retina (due to hemorrhage probably), ulcer of cornea, retinal arteriosclerosis, etc.

The subjective symptoms consist of the characteristic sequelae of these affections, *i.e.*, ambyopia, blurred vision, scotoma, inability to use the eyes without pain, hemianopsia, double vision, etc.

It will be observed that these symptoms, both objective and subjective, closely parallel those of albuminuria, with the exception of the cataract, and that in diabetes the stellate macular figure seen in albuminuria is absent, but differentiation is possible by other measures, as will be indicated in the proper place.

Concerning the relative frequency of certain ocular symptoms, no better information has been found by the writer than the following paragraph quoted from Knies¹:

"Among fifty-two diabetics, Lagrange (*Arch. d'Ophth.*, 1887, Jan.) found twelve cases of cataract and thirteen of hemorrhage into the interior of the eye. Among 144 diabetics, Galezowski (*Jahr. f. Aug.*, 1883, p. 297) observed five cases of paresis of accommodation, four of parenchymatous and purulent keratitis, seven of iritis, four of glaucoma, forty-six of cataract, twenty-seven of retinitis, thirty-one of amblyopia, ten of paralysis, four of hemianopsia, three of detachment of the retina and three of atrophy of the optic nerve. Among 169 diabetics, Mager (*Berl. kl. Woch.*, 1879, No. 21) found only five cases of cataract.

"It is evident, therefore, that the urine should be examined for sugar in every case of cataract which develops at an unusual period and without any known cause, as well as in every case of neuritis, retinitis, neuroretinitis and atrophy of the optic nerve, in every doubtful

1. Eye in General Disease, 439 *et seq.*

visual disorder, neuralgia or ocular paralysis. Hirschberg found diabetes in one per cent. of all his private patients."

Concerning retinal hemorrhages, Granclement¹ stated that he had observed thirty such cases, in all of which death occurred within eighteen months. In regard to the frequency of cataract, Uhthoff² appears to agree more nearly with Mager, as he states that five per cent of all cataract operations are performed upon diabetics.

Knies observes that the paralyzes of the ocular muscles are usually incomplete and transient. He gives paralysis of the ciliary as the most frequent and believes it to be especially suggestive when occurring in middle life. He mentions Gutman and Landesberg as having noted abducens paralysis; Kwiatowski, trochlear paralysis; Seegan, double ptosis; Galezowski, oculomotor paralysis; and Legrange and Ogle, facial paralysis, and states that these were due to nuclear and peripheral hemorrhages or peripheral neuritis.

The greatest optical effects produced by the disease consist of rapid changes in the refraction. These vary from minor changes to as high as 3.00 diopters, and they appear to be governed by no known rule, since, among the cases that have been presented, the difference in number between the cases displaying an *increase* in the refraction and those revealing a *decrease* is not sufficiently great to establish a firm basis for contention either way. Some authors, Gould,³ for instance, contend that an increase in the refraction is the rule, and that other changes should be looked at askance, while others, as is evidenced in the American Encyclopedia of Ophthalmology, apparently favor a decrease.

1. Lyon Med., January 16, 1910.

2. Prac. Med., Series 1910, page 100.

3. Biographic Clinics, Vol. VI.

Excellent arguments, supporting each hypothesis, have been advanced, but, in all probabilities, Wescott and Ellis¹ are right when they "call attention once more to the fact that the refraction not only may be increased in diabetes, but that it may be diminished; that both changes may occur in the same patient; and that the changes bear no necessary relation to the amount of sugar in the urine."

They summarize the arguments as follows:

"Numerous theories have been proposed in explanation of the refraction-changes in the course of diabetes, generally assuming that the presence of sugar in the lymphatic system of the eye produces alteration in the refractive indices of the media, cornea, aqueous, lens and vitreous; or else that the volume of the globe is altered through the loss of fluid in diabetes. For example, Landolt explained his case of diminished refraction on the theory of augmentation of the index of refraction of the vitreous body, since the vitreous presents a concave surface to incident rays. He thought that changes in the volume of the globe could not occur rapidly enough to account for the changes in refraction observed in his patient. On the contrary, Homer believed that sudden loss of fluid from the eye was responsible for developing hyperopia. Schmidt-Rimpler, not referring to diabetic cases especially, said that loss of fluid, with minus tension, does not alter the refraction of the eye. In perhaps the most thoroughly studied of all the cases found in literature, Lundsgaard's third case, repeated examinations with the ophthalmometer and the Schiotz tonometer revealed no alterations in corneal curvature or tension of the eyes during the period of varying refraction.

1. Journal American Medical Assn., August 12, 1911.

"An increase of refraction in the course of cataract development is a very frequent phenomenon. On the contrary, Macheck detailed three cases of diminished refraction in the course of the development of subcapsular cataract with central transparency of the lens; and Priestly Smith speaks of hyperopia developing in incipient cataract. In diabetic cataracts, the lens has been found, on chemical examination, to contain sugar (Liebreich Leber); Nagel, on the contrary, did not find sugar in the lens. Lichtenstein thought that changes in the index of refraction of the lens was the explanation of his case, as did Knapp, who compared the diabetic toxin with nicotine and strychnin in the rapidity of its action.

"Against the idea of alteration in the refractive index of the aqueous humor caused by the presence of sugar in this fluid, is the experiment of Hess, showing that twenty percent of grape sugar is necessary to produce a myopia of 1 D., while Deutchman noted cloudiness and swelling of a lens after immersing it in five per cent sugar solution for a few hours. Obviously these two experiments are inconsistent with the idea that a considerable change in refraction, without cataract formation, is due to the presence of sugar in the fluid bathing the lens.

"It seems to us none of these theories will do."

The authors cite a number of cases in substantiation of their claims that *the disease exercises no stable influence upon the refraction of the eye*, and the evidence they present certainly seems to confirm their conclusions.

It seems to the writer of this volume that there is virtue in *all* the claims presented. Their diversity merely tends to prove the diversity of the action of the disease.

For example, Landolt's theory that the increase in the index of refraction of the vitreous was the cause of diminished refraction in his case is perfectly reasonable and governed by that law of physics which decrees that a ray of light passing from a denser to a rarer medium is deflected away from the normal. It is a matter of course that the degree of deflection varies according to the difference between the media, growing less as the difference is diminished. If the index of refraction of the vitreous became the same as that of the crystalline lens, the posterior surface of the lens would lose practically its entire focusing power. In an eye otherwise normal, there would then exist hyperopia to the extent of the normal strength of the posterior surface of the lens.

It follows, then, that when the normal relationship between the vitreous and the lens is destroyed, there will exist a refractive defect in accordance with the extent of this destruction. Since it appears that in diabetes this destruction is due to the augmented value of refraction of the vitreous, the creation of hyperopia is to be expected.

On the other hand, there is also virtue in Zentmayer's¹ remark, "There is no other way in explaining the *increase* in the refraction except by a swelling of the lens."

Again, diminished refraction may well be due to paralysis of the ciliary muscle, thus giving a false increase to hyperopia or decrease to myopia, in much the same manner as a cycloplegic. It has already been stated that Knies found paralysees of the ciliary the most numerous of the ocular paralysees due to diabetes.

The writer has had little experience in such cases, the only one at all comparable with those mentioned in

1. Journal American Medical Assn., August 12, 1911.

medical literature being reported by him in the *Keystone Magazine of Optometry*, Jan. 14, 1915.¹ Even that case is not presented as one undeniably diabetic. The author well realizes that several of the points presented are debatable. Nevertheless, he is confident that the change in refraction from 1.12 D. hyperopia to .25 D. hyperopia inside of two months was due to this cause.

Gould's grounds of contending that there is always, or nearly so, an increase in the refraction in diabetes are rather unstable. Even his own case disproves his theory to some extent, since the refraction first increased beyond a point where it had stood for years, and then diminished below that point to a considerable extent.

The author's abbreviated presentation, in the *Keystone*, of Gould's case is given herewith:

"Patient, a physician, aged fifty-eight, was wearing, December 1902, and for some years previous:

O. D. —2.62 — .75 axis 35 V. A. 20-20

O. S. —2.62 —1.25 axis 167 V. A. 20-20

"On December 18 a marked change was felt by the patient, and an examination revealed the following:

O. D. —3.25 — .75 axis 25 V. A. 20-20

O. S. —3.25 —1.50 axis 166 V. A. 20-20

"Urinalysis indicated an excessive amount of sugar, and a strict diet was undertaken. In a few days all sugar traces were eliminated and refraction was found to have again altered to the original.

"Four years later — December 1906 — the patient having become careless in his diet, his defect amounted to

O. D. —2.87 — .87 axis 25.

O. S. —3.00 —1.50 axis 172.

1. Rapid Changes in Ocular Refraction.

"In February 1907, a strict diet was again undertaken, and in a few days, after the elimination of sugar, his refraction was

O. D. —2.00 — .62 axis 20.

O. S. —1.87 —1.37 axis 170.

"This correction gave freedom from all strain, and also perfect vision, and the author stated the condition would probably remain the same as long as no glycosuria was present."

In view of the author's justifiable contention that there could be no question as to the accuracy of all these refractions, the possibility of the final reduction in the myopia being due to presbyopia may be excluded. Presbyopic changes to such an extent (.87 D. in one eye and 1.12 D. in the other) are not likely to take place in the two months that intervened between the two refractions.

The evidence presented undeniably establishes diabetes as a causative factor in rapid changes in the ocular refraction. It therefore is obvious that when such changes occur, this cause may be among those sought. Since the number of causes is restricted, the process of elimination is comparatively simple. For instance, traumas and paralyses and spasms of the commoner type met with are usually accompanied by other symptoms which soon merit their exclusion from the diabetic class.

As previously stated, the subjective and objective symptoms closely resemble those of albuminuria, the more noteworthy exceptions being given, and the final analysis depends upon the condition of the urine and of the blood-pressure. With the former the optometrist

has little or nothing to do, tho that advance in optometric practice is expected by the author to come soon, but with the latter he may be vitally concerned. A blood-pressure corroborating other symptoms will go a long way toward establishing an intelligent understanding of the case.

CONDITION OF BLOOD-PRESSURE. Contrary to what may have been the expectation, the value of this symptom lies not so much in being characteristic as in being negative. The elevated pressure of albuminuria, which exists in such a high percentage of cases, may well be expected when ocular lesions are present. In the absence of this symptom, the cause of such lesions (hemorrhages, exudates, neuroses, pareses, etc.) may be sought elsewhere.

Janeway¹ substantiates this assertion in the following words:

"I have seen both high and low blood-pressure in diabetes; the latter in severe cases with marked emaciation and diacetic acid in the urine; the former especially in the milder forms in stout elderly people, where chronic nephritis or arteriosclerosis existed. I believe that the disease, of itself, is without influence on arterial pressure; that the occurrence of chronic nephritis, or arterio- or angiosclerosis, as a complication, explains the hypertension; and that the resultant emaciation, and the atrophy of fatty changes in the heart, cause the hypotension in severe cases."

E. H. Goodman has observed both high and low pressures, but the latter more often. He seems inclined to favor lowered pressure and states that "complications

1. Janeway, T. C.: The Clinical Study of Blood-Pressure.

are probably at the bottom of the hypertension, and only those cases which have nephritis or arteriosclerosis exhibit the phenomenon."

Geo. W. Morris¹ says, "Diabetes bears no constant relation to blood-pressure. In the latter stages hypotension may occur. Blood-pressure estimations may be of diagnostic value in differentiating between diabetic and uremic *coma*. Preceding the *onset of coma*, blood-pressure falls sometimes 40 to 50 mm. Hg. Blood-pressure readings are, therefore, of value in detecting the onset of coma."

The evidence in favor of a negative sphygmomanometric finding appears to be conclusive. But it must be remembered that it actually is this negative finding which constitutes a symptom of importance when ocular lesions as previously mentioned exist. Case 171, cited on page 216 of the final chapter, serves as a good example.

Nevertheless, the absence of an elevated or depressed pressure must not be accepted as conclusive proof either of the absence of albuminuria or of the presence of diabetes. A urinalysis and the other means at the physician's command must necessarily be employed to obtain a basis for a conclusive differentiation.

Chlorosis is a form of anemia often present in young women between the ages of fifteen to twenty-five; it is characterized by a greenish color of the skin, as the name would indicate, and menstrual disturbances.

The symptoms are so similar to those already enumerated under Anemia that they need not be repeated here.

CONDITION OF BLOOD-PRESSURE. As might be expected, this is low. Indeed, in all particulars, practically speaking, in the present consideration of

1. Blood-Pressure: Its Clinical Application.

chlorosis, the facts applying to anemia may be appropriately applied to chlorosis. Reference to that subject is recommended.

Exophthalmic Goitre, also known as BASEDOW'S DISEASE, GRAVES' DISEASE, FLAJANI'S DISEASE, HYPERTHYROIDISM. While this disease is not one intruding into optometric practice with great frequency, its cardinal symptoms include ocular phenomena of sufficient import to justify presentation.

As the final name would indicate, the disease is characterized, in a great majority of instances, by an enlargement of the thyroid glands, situated in the neck, adjacent to the windpipe. This symptom, combined with tachycardia and exophthalmos, completes the triad of cardinal symptoms of which one or more may be expected in every case of the disease. To quote from the American Encyclopedia of Ophthalmology: "Of the three cardinal symptoms, tachycardia is almost constant, and is the first to appear. One or both of the other symptoms may be absent in about one-tenth of the cases and goitre in about one-twelfth. The heart's action always reaches 100 per minute, and has been frequently noted at 200."

Aside from the exophthalmos, *per se*, there are several less spectacular symptoms of note, constituting *signs* which aid in an intelligent understanding of the case. Of these, Von Graefe's sign is perhaps the most notable. It consists of the failure of the upper lid to follow the eye in its rotation downward. This lack of proper coordination results in there becoming exposed an area of the sclerotic *above* the cornea when vision is directed downward, clearly a condition of abnormality, as is at once evident, since the lid normally follows and conceals the upper section of the cornea and iris.

This sign has been found in variable degree, both numerically and mechanically. In the former sense, its frequency, as given by various authors, ranges from fourteen per cent to ninety-eight per cent. In the latter sense, the extent of departure from normal necessarily varies according to the progress of the disease. Generally speaking, it will become more pronounced as the disease progresses.

Dalrymple's sign consists of a retraction of the upper lid *at all times*, in contradistinction to Von Graefe's sign in which there is a relative retraction only when the eye is directed downward. The effect produced is a staring and apprehensive expression. This sign is not to be depended upon, however, as it results from other conditions as well; namely, amaurosis, orbital tumor, some forms of hysteria, tetanus, stimulation of the cervical sympathetic, etc.¹ Aneurysm, also, as described on page 91, could produce a similar result.

In cases where the protrusion of the eye is very pronounced, there will exist an inability to completely close the lids. The resulting non-lubrication of the cornea produces disastrous results, as can readily be imagined.

Möbius' sign consists of the inability of the eyes to converge for near vision.

Von Stellwag's sign consists of lessened frequency of nictation. The act is also imperfectly performed, there sometimes occurring a rapid succession of winks, and then a considerable pause, and frequently, or perhaps invariably, the lids do not meet when the act is performed.

Joffroy observed that when the patient inclined the head downward and elevated the visual axis, the fore-

1. American Encyclopedia of Ophthalmology, page 4807.

head did not wrinkle as it would in a person free from exophthalmic goitre.

It appears to be generally conceded that the cause for these various phenomena may be attributed to sympathetic stimulation of Mueller's orbital muscle, the resulting contraction of the muscle pulling the eye forward, thereby mechanically preventing normal action of the lids. The abnormal rigidity, too, thereby imposed upon the ball would necessarily tend to prevent free action of the internal rectus (Möbius' sign.)

In addition to this factor, it is also believed that the cause of the exophthalmos is at least partially due to an increase in the orbital contents. It has apparently been clearly demonstrated that there is an increase in the orbital fat, and no doubt the excessive muscular action tends to increase the blood supply. The tendency of the exophthalmos to diminish after death encourages this hypothesis.

As for other eye symptoms, Knies mentions dilatation of the pupils, sometimes unequal; tremor of the eye lids, and nystagmus; keratitis, owing to the exposed cornea; pulsation of the retinal arteries; rarely, optic neuritis and concentric narrowing of the field of vision; muscular paralysis, involving the trochlearis, abducens and motor oculi, also the orbicularis, trigeminus, facial, glosso-pharyngeal, spinal accessory and hypoglossal nerves. Color blindness, paralysis of the extremities, epilepsy and loss of smell and taste have been reported.

Of course, most of the disorders arise from toxemia. Some, also, are initiated by coincident diseases, such as diabetes, nephritis, syphilis, etc.

When the orbital muscles are involved, it is natural that subjective symptoms will occur. These sometimes

consist of headache, vertigo, retinal and muscular asthenopia and other symptoms of pure eye-strain. The severity of other symptoms, however, will soon permit of differentiation.

CONDITION OF BLOOD-PRESSURE. This symptom is variable. It has been mentioned that arterial pulsation has been observed a number of times, and it consequently follows that the extraocular arterial pressure must be low, at least lower than the intraocular pressure, since the blood was forced into the eye only during the period of systole. In such cases it is quite possible that the toxic products of hyperthyroidism have paralyzed the vasoconstrictor nerves and thereby permitted an abnormal distention of the arterial system. It follows that if the distention obtains in the entire system, there will be a general lowering of both diastolic and systolic pressure. If only the smaller, peripheral arteries are involved, there would exist a normal, or perhaps high, systolic and a low diastolic, owing to the sudden transference of blood from the arteries to the capillaries and veins; in other words, to the lessened peripheral resistance. The pulse pressure would therefore be high.

On the other hand, it is not impossible that in the initial stages, at least, the toxemia results in a *stimulation* of the vasoconstrictor nerves, with a concomitant *rise* in blood-pressure. The state of arterial tension existing under such circumstances would be high systolic, high diastolic, low pulse pressure, almost a reverse of the previously cited condition.

Hirschfelder¹ has found the maximal blood-pressure usually to be high, and the minimal normal, the pulse pressure being increased. Yet, if, as he says, the figures

1. Diseases of the Heart and Aorta, page 687.

he gives, which are the findings of Krause and Frieden-thal,¹ represent the pressures he has found to exist, it would seem that the elevation is not very great, as such a condition is generally understood. The figures given correspond very well with the suggested pressures in a case of peripheral vasomotor paralysis—a normal or high (slightly) systolic, low diastolic and high pulse pressure. There is a relatively low diastolic in all the figures given.

The same writer says, also, that the maximal pressure may not be elevated even when there is tachycardia. It follows, then, that in view of the general uncertainty concerning blood-pressure, it is a symptom not to be regarded as of consequence in the diagnosis of exophthalmic goitre.

I.	Lowest	Average	Highest
	Mm. Hg.		
Max.	106	134	158
Min.	62	65.8	66

CHAPTER 10.

SYPHILIS

The frequent mention of this disease in medical and allied literature has been referred to (*vide* chapter on Arterial Pathology). Because of the migrating propensities of the characteristic germ of syphilis (*Spirochaeta pallida*), no portion of the body is safe from an invasion by it, and, in consequence, every specialist finds syphilis occupying a dominant position in the particular field of pathology with which he is concerned.

Those engaged in the various forms of eye-work are certainly not excepted, as the disease all too often proves an antagonist ever in the foreground, and one not to be conquered by local attacks. General treatment is usually called for.

As previously stated, paralysis of the third nerve is believed to be almost pathognomonic of syphilis. Certainly the association of the two conditions is remarkably frequent; so, also, is the occurrence of other muscle paralyses with syphilis. The writer has found paralysis of the trochlear nerve not infrequently, and that of the abducens less infrequent, altho, on the whole, there has been little choice between them.

The relative frequency of the three forms of palsy has therefore not been found as unequal by the writer as by

Knies, who rates them as follows: about seventy-four per cent oculomotor; about twenty-four per cent abducens; and one per cent or two per cent trochlear. The writer is, of course, obliged to defer to the far greater knowledge and experience in this subject of the author mentioned. It would be presumptuous to assume otherwise. Nevertheless, that others *apparently* have found it necessary to differ with Knies is evident from the following statement by Hansell and Reber¹:

"The long course of the sixth nerve at the base of the brain renders it peculiarly liable to arrest of function during disease, or from traumatism to the skull. Indeed, the external rectus leads in the order of frequency of individual ocular palsies, the order being external rectus first, then unilateral oculomotor palsy, paralysis of the superior oblique, the inferior rectus, the superior rectus, the internal rectus and the inferior oblique. Duane (Knapp's Arch. of Ophthal., 1894, p. 61) would award to the superior rectus second instead of third place. Convergent strabismus will be simulated as the eyes are moved toward the palsied side.

"Abducens palsy is commonly seen after fractures, or accompanying a syphilitic or other inflammatory process at the base. It also occurs with diseases of the pons. Peripheral or orbital abducens palsy, when not rheumatic in character, is said by Wood to indicate syphilis in adults and tubercular disease in children."

However, this quotation relates more to the relative frequency of the individual paralyses rather than to the frequency of the *causes* of them, and so it still leaves room for argument.

1. Ocular Muscles, page 62.

There can be no argument, however, regarding the great part syphilis takes in causing ocular paralyses. According to Alexander,¹ 59.4 per cent of all ocular palsies are specific. He believed the oculomotor led in frequency.

Knies thought that "perhaps a half of all paralyses of the ocular muscles are syphilitic in origin. Indeed, every spontaneous ocular paralysis or ophthalmoplegia necessitates a differential diagnosis with regard to syphilis."

All degrees of muscle anomalies may arise, ranging from a *tendency* to deviation to an actual one. As intimated in the above quotation, parallelism may exist while the eyes are directed to one position, and convergence (or divergence) occur when directed to the opposite one. When the anomaly is not too pronounced, the writer has found the application of prisms gratefully accepted by the patient. They serve not only to allay subjective symptoms, such as nausea and diplopia, but also to relieve an impaired neural and muscular system, and, to that extent, aid in its recuperation.

These prisms are to be worn temporarily or constantly, according to the manner in which the local affection yields to the general treatment. If there is a reduction in the amount of the anomaly, it is necessary, of course, that the prisms be reduced if they tend to over-correct. Within reason, the same method should be followed in case of an increase in the defect, always bearing in mind, however, that prisms over five degrees in strength are practically never feasible. Naturally, if the defect entirely disappears, the prisms are eliminated; if it greatly increases, and remains permanent, operative measures may be indicated. Such cases should be placed under the supervision of the ophthalmic surgeon, if the attending physician sees fit.

1. Syphilis und Auge.

Prognosis in such cases is only fairly encouraging. According to Naunym (Knies) seventy per cent recover. Hansell and Reber have found that much depends upon the character of the affection—whether acute or chronic, individual or associated, traumatic or idiopathic or due to central or peripheral lesion. Altogether, the appearance of the anomaly calls for careful and thorough medical attention. Collins and Wilde¹ investigated 120 cases and found the results as follows: recoveries twenty-four per cent; marked improvement sixteen per cent; death twenty-four per cent.

Paralyses of the internal ocular muscles are not particularly rare. The affection may be either unilateral or bilateral, but the former has been found more frequently by the writer than the latter. The condition is manifested by an inability to accommodate for near vision. When unilateral, it is very striking. Knies mentions only the unilateral variety and states that Alexander believed three-fourths of such cases were syphilitic, while Uthoff thought only one-fourth were.

Syphilitic paralyses of the eye muscles occur in the latter stage of the disease, after six months or so. Naunym expressed the opinion that there was no hope of recovery if evidence of improvement did not take place within two weeks.

Iritis may be attributed to syphilis in many cases. Indeed, some writers assert that syphilis is the most prolific cause of this ocular affection. It occurs at an early stage and is at first unilateral, becoming bilateral if not checked.

In the beginning it is said to be not different from the plastic iritis due to other causes. When characteristic,

1. American Journal Medical Science, November, 1891.

it appears as yellow or dirty orange colored nodules in the tissues of the iris, these rarely being more than two-thirds of a millimeter in diameter and surrounded by a narrow red zone. They are usually situated upon the border of the pupil. For other information upon this important subject the reader is referred to standard works upon ocular diseases. Iritis, when it occurs, is so painfully evident that extensive comment upon it is considered unnecessary in this work.

Choroiditis is frequently caused by syphilis—either congenital or acquired. The American Encyclopedia of Ophthalmology¹ gives syphilis first place among the causes of *disseminated* choroiditis in its several forms by stating it to be the chief cause. Parsons² concurs in this opinion. So, also, do Knies and Noyes.

The usual fundus picture is presented in such cases, the ophthalmoscope revealing comparatively small whitish areas usually surrounded by black borders or rings of pigment quite similar in character to the choroidal crescents bordering the nerve-head in many cases of myopia, etc. Occasionally these areas of degeneration, which usually assume an elliptical form, contain isolated masses of black pigment; at other stages of the disease, they may be entirely black.

The subjective symptoms of the disease may be reduced vision, due to cloudy vitreous or destroyed retinal tissue; distortion of images, resulting in irregularity of appearance; micropsia and macropsia. The latter three symptoms are due respectively to an unevenness of the underlying diseased choroid which irregularly raises the

1. Page 2141.

2. Page 358.

overlying retina; to a separation, and to a crowding together of the rods and cones from the same cause.

Other sequelae of an irritated retina may be present, such as colored vision, scotoma, flashes of light, etc. In fact, many of the subjective and objective symptoms of retinitis may exist in syphilis. Parsons states that syphilis is one of the most common causes of retinitis, the lesion usually being secondary to an accompanying choro-oiditis.

In arteritis obliterans the small arteries of the retina become narrow and finally occluded. The significance of this lies in the fact that the retinal arteries are peripheral arteries, and when they are so affected it is not unlikely that others are likewise affected. As the cerebral arteries come into this category, it is natural to suspect that certain muscular or visual abnormalities, due to disturbed cerebral vascularization, may be exhibited in such cases. It is apparent, then, that the arteries of the retina may present evidence of the utmost importance in regard to disturbances of the central nervous system, since, of course, organs other than the eye may be affected.

Floating opacities in the vitreous may be present in syphilis, tho, of course, they may be absent, or present in other forms of ocular disease.

Keratitis, as a result of congenital syphilis, is not at all uncommon. It seems to be more or less of a periodical nature, and may be most severe, intolerable photophobia being present. As a result of acquired syphilis, it is said to be rare. The congenital form usually attacks girls between the ages of five and fifteen (Parsons). Associated with this symptom frequently are the so-called

Hutchinson teeth. The irregularity of this form of tooth is to be noted in the accompanying diagrams.

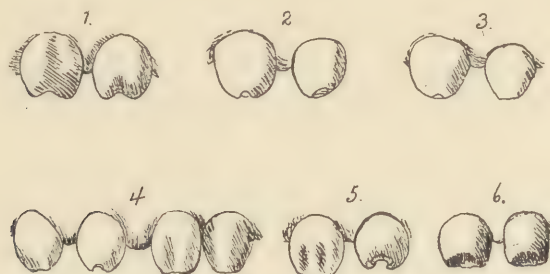


Fig. 17

Hutchinson's Teeth (Nettleship)

It will be observed that these teeth (the upper central incisors) possess the following characteristics: Notching of the central cutting edge (1-6); peg, or screwdriver, shape (1, 2, 3); diminutiveness (2, 3 right); separation (chiefly because of reduction in size). The left tooth at 5 is practically normal.]

It is almost a matter of course that the optic nerve is affected in syphilis, especially when cerebral lesions occur. The affected area may be confined to that part of the nerve which is situated in the globe or it may exist at any point along the course of the nerve to the brain. The former condition is known as *optic neuritis*, and the latter as *retro-bulbar neuritis*.

The ophthalmoscopic evidence may vary from barely perceptible hyperemia of the nerve-head to the spectacular appearance of a choked disk. In view of the many variations of the normal optic disk, the former is very difficult to distinguish with certainty in many cases. Difference in the degree of its development upon the two nerve-heads may aid in arriving at correct conclusions. Particularly the definition of the edges of the disk serve this purpose. When they are blurred and indistinct in outline, merging perceptibly with the surrounding retina, diagnosis is rendered much easier.

The swelling of the disk is likewise an unmistakable sign. The extent of the elevation of the nerve-head is, of course, variable; sometimes, in extreme cases, it is as much as 8-10 D. (Parsons).

It seems hardly necessary to mention, however, that extreme papillitis is caused more often by brain tumor than syphilis, being regarded more or less as pathognomonic of the former, since the tumor exists in about eighty per cent of cases. Nevertheless, Parsons gives syphilis as one of the causes.

Badal (*Arch. d'Ophthalm.*, VI, p. 301) found 631 cases of ocular syphilis to present 139 diseased optic nerves. He also found among them 144 muscle paralyses.

Prognosis, it seems, is not particularly good, as the final result depends upon whether or not the disease in general is halted. This phase of the case rests entirely in the hands of the attending physician.

CONDITION OF BLOOD-PRESSURE. A study of contemporaneous medical literature leads to the conclusion that the blood-pressure in syphilis is a very uncertain factor, being dependent upon the extent of the affection and its principal seat of attack. Thus, as an etiologic factor in aortic regurgitation (*q.v.*) it may result in a high systolic pressure, accompanied by a low diastolic. As a cause of arteriosclerosis, there may be expected blood-pressure sequelae as indicated in the chapter on that pathologic condition, *i.e.*, elevated pressure in the primary stages, when the vessel walls are hardened, but not materially stretched, and lowered pressure in the secondary or tertiary stages, when the constant pressure has permanently widened the lumen of the vessels.

As a matter of fact, syphilis enters very largely into pathology of the circulatory system and, in the present connection, that chapter may profitably be studied in conjunction with this one.

Hirschfelder¹ mentions the tabulation by Pearce of several investigations in which it is conclusively demonstrated that about thirty per cent of a large number of cases of various forms of arteriosclerosis gave a positive reaction to the Wassermann test. It is obvious that each form will, if uncomplicated, yield blood-pressure characteristic of it. For example, aneurysm of the larger vessels² results (probably) in lowered systolic pressure, the diastolic remaining the same unless the dilatation is large enough to hold a quantity of blood sufficient to lessen pressure over the entire vascular system. It is in such cases that pulsation of the retinal arteries might occur. As previously stated, there is no characteristic blood-pressure finding in aneurysm. The condition is one entering too rarely into optometric practice to merit elaboration.

1. Page 341.

2. "The rôle of syphilis (as a causative factor of aneurysm) was demonstrated definitely by Lancisi (1728)," Hirschfelder, page 619. The same author says, "In communities where syphilis is common the frequency of aneurysm increases. Thus, it is eleven times more common in the British Army in India than in the civilians at home, and much more common in the British than in the Austrian and German Armies, where venereal disease is five times less prevalent." Statistics indicate aortic aneurysm to be eleven times as frequent in men as in women. There is a much more even percentage in other forms.

CHAPTER II.

TOBACCO

Tobacco. Toxic amblyopia appears to be the most pronounced ocular effect resulting from excessive use of tobacco. While the condition as the result of tobacco alone is rare, its occurrence when the deleterious effect of alcohol is combined with that of nicotine is relatively frequent. The following table, taken from Noyes' "Diseases of the Eye," page 683, gives interesting information in this respect. The figures relate to the causes of retrobulbar neuritis in 204 cases in which that affection was found.

From abuse of alcohol.....	64	From syphilis, acquired.....	7
" alcohol and tobacco.....	45	" hereditary.....	7
" abuse of tobacco.....	23	" multiple sclerosis.....	5
" diabetes.....	3	" cold.....	5
" lead.....	1	" menstrual disturbances.....	3
" sulphuret of carbon.....	2	" pregnancy.....	4
		" loss of blood at abortion.....	2
	138	" anomaly of heart.....	1
		" periostitis orbitac.....	1
		" unknown.....	31

It will be observed that the frequency of tobacco poisoning is only about one-third that of alcohol poisoning and one-half that of the two combined. However, it is not unequivocally stated by the author whether or not the sixty-four cases due to abuse of alcohol abstained *entirely* from tobacco. It is difficult to conceive of individuals addicted to the abuse of alcohol refraining entirely from

the use of tobacco, and this fact inclines the writer to the belief that tobacco poisoning entered to some extent into the cases mentioned¹; there probably, however, did not exist the *abuse* in these cases that made the others notable.

The condition is due to an inflammation of the axial fibers of the nerve by the poison, and their subsequent

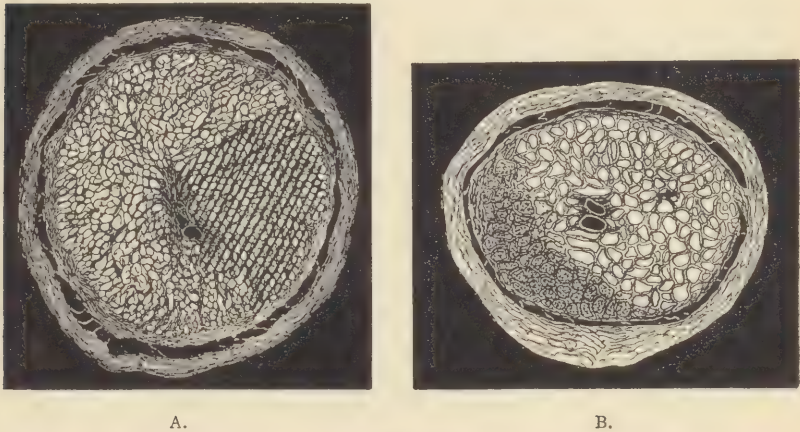


Fig. 18

A, Merkle's diagram showing relative position of macular fibers just previous to their entrance into the right eyeball. B, Modification of Uhthoff's figure showing degeneration of infero-temporal fibers due to nicotine and alcohol. Section taken just behind the left eyeball.

atrophy. This final stage is revealed by the ophthalmoscope as a pallor of the optic disk in the region of the infero-temporal quadrant as is indicated by Fig. 18, B. Since the fibers from this area supply the macular region, the amblyopia is usually central or paracentral. Yet this one objective symptom may not be regarded as pathognomonic unless accompanied, not only by central amblyopia, but by a relative central scotoma for color, preferably and commonly red and green, since it may be

1. Nettleship has observed that he never saw a case of *pure* alcohol amblyopia.

due to other causes. Myosis, also due to the effect of nicotine upon the pupil contracting-center, in contradistinction to the mydriatic effect of alcohol, may be expected.

The detection of the color scotoma may be easily accomplished by looping a piece of red or green yarn and allowing a quarter inch of the loop to project beyond the finger and thumb. Seating himself at a distance of about two feet from the patient, who has one eye effectively obstructed by a bandage or similar contrivance, the examiner directs the patient to look steadily with the unobscured eye into the pupil of his (the examiner's) eye. He now interposes the loop of color into the mutual line of vision about five or six inches from his own eye. If tobacco or alcohol amblyopia exists, the patient will not see the loop as a color, but will require that it be moved an inch or two to either side or up or down, before such recognition will take place. When this change of position is made, the loop will assume its natural color, or, if the eye's central color sense has not been entirely lost, it will become more intense in shade.

Knies states that "tobacco amblyopia rarely develops in a strong, youthful individual. It develops either at an advanced age, when the resistance to external agents has diminished, or when the general nutrition has been impaired by anorexia, chronic gastric catarrh, etc."

Other local affections resulting from the use of tobacco are too rare to deserve space here. Disturbances of muscles other than the sphincter iridii are questionable. While it is not the province of this work to speak of corrective measures, it is justifiable to depart from the rule and state that total abstinence is necessary if relief is desired.

CONDITION OF BLOOD-PRESSURE. Owing to the vaso-constriction value of nicotine, its immediate effect is to raise the blood-pressure; this represents the *acute* phase of the poisoning. The *chronic* phase (habitual users), in which there is repeated inhalation or deglutition of nicotine, appears generally believed to be represented by a lower pressure which may be due to arterial sclerosis.¹

The use and abuse of tobacco have become so common that more than passing mention is deserved, and so the following quotation from Hirschfelder² is given, that the viewpoint of the highest authority may be understood:

"Lee found that in non-smokers the first effect of smoking a cigar was to produce a rise of 10-20 mm. Hg. in the maximal blood-pressure, which was often associated with palpitation. Within five minutes after this the maximal blood-pressure fell 50 mm. Hg., and this fall was accompanied by pallor, sweating, weakness, and colicky pains in the abdomen, as well as by the appearance of *muscae volitantes*, irregularity and weakness of the pulse,—or, in other words, the symptom complex of arterial anaemia.

"In more habitual smokers, those of the second group, a single cigar produced only the rise of blood-pressure and palpitation. The subacute symptoms, therefore, come on only as the result of excessive indulgence.

"In old habitual smokers, these observers found either no effect whatever, or only a slight rise of pressure resulting from a strong cigar, without any of the disagreeable symptoms.

"Lee's observations have been repeated by Bruce, Miller and Hooker, who found that smoking increases the

1. The writer recently observed two cases in which there was pronounced excessive use of tobacco. The first, a man of about 35, smoked 12 to 20 cigars a day and had the following pressures: Systolic 90 mm., diastolic 60 mm., pulse 30 mm. The second, a man of 53, smoked 20 to 25 cigars a day and had pressures as follows: Systolic 95 mm., diastolic 70 mm., pulse 25 mm.

2. Diseases of the Heart and Aorta, page 714.

maximal, minimal, and pulse pressures in man, tho later these return to normal. The cardiac output, therefore, seems to be increased at first, as Lee had found in cats. Bruce, Miller, and Hooker also found that the volume of the hand always decreased during smoking (vaso-constriction), whereas Lee found that the volume of the cat's intestines also decreased. It is probable that a little later there occurs in man a dilatation of the abdominal vessels, but it is not yet certain that it does so.

"The chief sufferers from tobacco are the young cigarette smokers, who inhale the smoke, and these soon suffer immediately from the physiological effects of the nicotine. They complain of weakness, giddiness, intense palpitation and tachycardia (from continued stimulation of the cervical ganglion cells), and often of irregularity of the heart, which may be very distressing. It is most noticeable that the intense sensory disturbances occur without any motor insufficiency of the heart. Thus, a young man of twenty years, an habitual inhaler of cigarette smoke, recently consulted the writer, complaining of fatigue, giddiness, *muscae volitantes*, intense palpitation, but, on further questioning, stated that he was in the habit of running a quarter of a mile every evening for exercise, and after this exercise he had neither palpitation nor shortness of breath! Needless to say, he improved at once after stopping tobacco."

Sir Lauder Brunton¹ has observed that he knew of nothing, unless it be extract of the suprarenal capsules, which causes such a tremendous contraction of the vessels and raises blood-pressure to such an enormous extent as does nicotine when it is injected intravenously.

1. Brunton's Lectures on the Action of Medicine.

CHAPTER 12.

ALCOHOL

Alcohol. "It will perhaps suffice to say here that amblyopia from tobacco alone differs in no essential from that set up by alcohol alone, or by alcohol plus tobacco, either in symptoms, the character of optic nerve lesion, or in its treatment." This remark, taken from the American Encyclopedia of Ophthalmology, page 303, renders unnecessary a repetition of the information presented under Tobacco in so far as it pertains to amblyopia.

The frequency with which retrobulbar neuritis and its sequelae occur may be of interest, however. Uhthoff gives the results of an extensive investigation among 1,000 cases of "alcoholismus." Among these, 6 per cent were affected with amblyopia; in 6.5 per cent more he found the peculiar nerve lesion *without* amblyopia; and in 5.3 per cent, pathological conditions of the nerve and adjacent retina. Other lesions affecting the pupil, retina, and muscles brought the total percentage of cases affected with ocular diseases to thirty per cent!

Among the amblyopic cases, the ophthalmoscope revealed atrophic pallor of the temporal side of the nerve in sixty-three per cent; a slight but distinct haziness of the nerve-head in eight per cent, and in twenty-eight per cent no abnormal appearance.

It may be mentioned *en passant* that many ocular lesions more spectacular than those mentioned may be

indirectly due to alcoholism. Note 1, on page 238 *et seq.*, effectively points out that even moderate indulgence in alcoholic drinks brings its inevitable result, and it is quite in line with reason to conclude that in many cases the habitual indulgence in small quantities of alcohol eventually leads to renal and vascular diseases which ultimately include extensive ocular changes, such as retinitis hemorrhagica and albuminurica, thrombosis, etc., in their termination. These cases are classed as due to renal disease, but in truth they may be due, fundamentally, to other causes.

While it has been stated (Noyes, etc.) that absolute blindness due to alcohol is extremely rare,¹ central vision may be reduced to 1-10 or less (Knies). To a lesser extent the same holds true of tobacco amblyopia. The American Encyclopedia of Ophthalmology gives the reduction of vision in the latter case as at least 2-5 or 2-7 of normal. Concerning the distinguishing points between tobacco and alcoholic amblyopia, it has been said (Hirschberg) that in the former, scotoma is situated near, but not at, the point of fixation (paracentral), while in the latter it always *includes* the center of fixation. As previously stated, in nicotine poisoning the pupil is likely to be contracted, while it will be dilated in alcoholic poisoning. In the former there may be an associated spasm of accommodation, and Noyes says that muscular asthenopia is common.

True muscle paralyses are said to be rare. Uhthoff found only three in one thousand cases, each of which was a double abducens paralysis. Of course, the temporary paralyses resulting from *acute* alcoholic poisoning (intoxication) are too well known to merit other than passing comment.

1. Of course, imbibition of methyl (wood) alcohol is not considered in this connection.

CONDITION OF BLOOD-PRESSURE. The vasodilatation value of alcohol causes a lowering of the arterial tension. The action of the drug appears to be greatest upon the vessels lying in the skin. In some quarters it is further believed that chronic poisoning by alcohol (habitual drinkers of comparatively small quantities) results in an arteriosclerosis, which, consequently, elevates the arterial tension; in other quarters this is disputed. Cabot, for instance, found that the imbibition of large quantities of alcoholic drinks seemed to exert no influence in producing arterial disease.

Hultgen studied 460 cases of chronic alcoholism, and his deduction substantially verified those of Cabot. Fahr performed 309 autopsies on habitual drunkards dying at the Harbor Hospital of Hamburg, and of these the number of cases possessing arteriosclerosis was no higher than in patients who drank no spirituous liquors.

On the other hand, Aubertin caused arteriosclerotic changes in rabbits by the injection of alcohol; also cardiac hypertrophy. Thayer and Brush, in a study of several thousand cases admitted to Johns Hopkins Hospital, in which sclerosed arteries were diagnosed, found alcohol an etiological factor in 46.8 per cent. However, as Hirschfelder has so pertinently pointed out, it is difficult, if not quite impossible, to state unequivocally the cause of the disease in a given case, as many other factors may be contributory. Manual labor, for example, is a very frequent cause of sclerosis, and as a high percentage of those so engaged imbibe more or less alcohol, it would be impossible to state which exerted the greater influence.

The same author,¹ however, writes as follows:

"Fortunately, however, for the decision of these doubtful points, the experiments of Pic and Bonnamour

1. Diseases of the Heart and Aorta, page 341.

(*l.c.*) upon experimental adrenalin arteriosclerosis has shown that where two factors are acting together, arteriosclerosis may be produced in conditions in which it could not be brought about by one of them alone. Thus, tuberculosis and adrenalin yielded arteriosclerosis in young rabbits which would not have shown arteriosclerosis after adrenalin alone, and there is no doubt that the same is true in man."

It can readily be appreciated that if alcohol *is* a cause of arteriosclerosis, while the primary stage will cause a lowering of the blood-pressure, the secondary one, once the arteriosclerotic condition is attained, will show an elevated pressure. Then, too, it should not be forgotten that alcohol is often a factor in kidney and liver diseases and that diseases of these organs tend to raise the blood pressure.

Regardless of the merits of the controversy as above presented, the general deleterious effect of alcohol is too well known to permit a technical dispute to lessen the wisdom of at least moderate, if not total, abstinence. The facts presented in the appended note on page 238 establish beyond the shadow of a doubt the advisability of such a course.

CHAPTER 13.

ATROPINE

Atropine, being a drug frequently used in ophthalmological work, and therefore brought more or less to the attention of practitioners of optometry, deserves more than passing attention.

The typical effect of this drug upon the eye is well known. The writer feels that he can do no better, in presenting information concerning it, than to quote as follows from the American Encyclopedia of Ophthalmology, VOL. I, page 673 *et seq*:

“Atropia is a powerful cycloplegic and mydriatic and may act as a poison even when instilled into the conjunctival sac. In toxic doses it causes dryness of throat, vertigo, frequent pulse, a scarlatinous rash, hallucinations and delirium. A single drop of a one per cent solution of atropia sulphate produces mydriasis in about fifteen minutes, and cycloplegia in two hours. It stimulates the radiating muscular and sympathetic fibers of the iris while it paralyzes the peripheral cells of the motor oculi supplied both to the iris and to the ciliary muscle. Instilled thrice daily for two days, the cycloplegia lasts from seven to twelve days—the dilatation of the pupil somewhat longer. Atropia also increases the intraocular tension, and this fact should always be borne in mind in cases of suspected glaucoma.”

Of the latter effect the same work says:

"The most noteworthy toxic effect of the instillation of atropine is glaucoma. A large number of recorded cases show that a single atropine instillation may not only induce an acute attack in a latent, chronic glaucoma, but may set up in apparently normal eyes the same symptom-complex. Some observers assume that such eyes could not have been normal, that at least a definite predisposition must have been present, and that in healthy eyes atropine does not produce such serious changes. Perhaps some of the early observations of blindness following the use of belladonna or atropine can be explained by its production of acute glaucoma.

"Of thirty-two cases of glaucoma collected by Miles Standish, nine were believed to be due to atropin, two to homatropin, and one each to scopolamin, duboisin and cocaine (Ophthalmic Record, 1902, p. 243).

"No age seems to be exempt from such a possibility. The collected data show that children from five to ten years, as well as adult men and women, may be subject to this trouble. The climacteric period is said to predispose to it; likewise senile changes in the arterial system have been supposed to contribute to the rise of intraocular pressure produced by atropin as well as by glaucoma, but incorrectly. The strength of the atropin solution seems to be unimportant, for a solution of one to 2,500 may produce glaucoma as well as a solution of one to 100. The time required for the appearance of the glaucoma is variable. In one case it appeared in half an hour; in others in one, two, three and five hours, or even after several days."

Concerning its effect upon the general system the following is given:

"A too liberal application of atropin or its related poisons will sometimes produce symptoms that show themselves occasionally after a few minutes. These are chiefly dryness of throat, hoarseness, difficulty in swallowing, deep redness of the face, and a scarlatina-like eruption. In addition there is noticed quickening of the pulse, headache, vertigo, and delirium of a peculiar, restless, 'merry' variety. In one case, Raphael observed (1889) glycosuria, which disappeared with the other symptoms.

"If the dose is a fatal one (statistics of lethal doses vary greatly) death takes place in from two to twenty hours, preceded by violent quickening of the pulse and respiration, coma and paralysis of the sphincter muscles of the bladder and rectum."

Parsons¹ has the following to say regarding atropine:

"It paralyzes the sphincter iridis and ciliary muscle completely, and is said also to stimulate the dilatator iridis. It has so potent an action that it abolishes the tone of the ciliary muscle. Thus, an emmetropic eye placed fully under the influence of atropin becomes hypermetropic to the extent of about 1 D.; this must be taken into account in correcting errors of refraction."

Noyes² says, "If toxic effects appear, they will come as dryness of the fauces, which need not be heeded; but more important are, quickening and weakness of the pulse, flushing of the face, palpitation of the heart, headache, nausea, prostration, garrulous delirium, desire to urinate, and sometimes muscular violence."

Knies³ points out that "the most acute symptoms of poisoning have followed the introduction of a single drop

1. Diseases of the Eye, page 73.

2. Diseases of the Eye, page 240.

3. *Loc. cit.*, page 33.

of the ordinary solution," and mentions glaucoma, conjunctival catarrh, eczema of lids, visual hallucinations, difficult deglutition and epistaxis as reported consequences.

The medicinal value of the drug in iritis and other forms of disease¹ is unquestioned; however, its value as an aid to accurate refractive work will probably always remain a much mooted point. In view of the fact that the refractive work of those who do *not* use it is at least equal to that of those who do, there hardly seems to remain a logical reason for its continuance in this class of eye work. Especially is this true when there exists the possibility of its instillation into the conjunctival sac causing the symptoms of toxemia above presented.

The writer, as well as many others, has demonstrated in thousands of cases that atropine is non-essential in the accurate ascertainment of the refraction of the eye. His experience has proven, at least to his own satisfaction, that ciliary spasms yield readily in practically all cases to modern refractive methods, and that a cycloplegic is absolutely uncalled for.

Regarding the permanence of the effect of atropine upon the ciliary muscle, as far as the writer is aware there has been no conclusive proof offered either by the proponents or the opponents of the theory that such an effect is produced. The claim which the writer heard Dr. Louis Wessels, of Philadelphia, make to the effect that in several thousand cases of atropinized eyes in children he had observed no permanent deleterious effect in a single case can hardly be accepted as scientific proof, inasmuch as a systematic investigation of a certain number of given cases through a period of, say, ten years did not appear to have been made.

1. For example, it is used as a heart accelerator, since its action is to paralyze the functioning power of the vagus nerve, which is the inhibiting nerve of the heart.

On the other hand, neither can the very able article by Mr. Geo. Rogers, of Chicago,¹ arguing to the contrary, be accepted as scientific proof, even granting the sound basis from which he reasons, since he has advanced a *theory*, not a sufficiently proven *fact*.

It appears to the writer that there is one way in which a legitimate conclusion can be arrived at, and that is to take two hundred or more cases, selected for their freedom from inherited or acquired diseases which affect the eyes, and for the very commonplaceness of their ocular condition; their ages to be approximately thirty, since their amplitude of accommodation before that period of life is too great and elastic to permit of accurate measurements. Let one hundred of these cases be examined *under atropine* every three years for twelve years, and the amplitude of accommodation taken in each case *before and after* each examination. Let the other one hundred be examined in a similar manner *without atropine*. Then at the end of twelve years, cast up the results. The writer is morally certain it will be found that the atropinized eyes will have a lessened average accommodative power.

In support of this final assertion, he wishes to point out that it has been his experience that there rarely is necessity for bifocals, or their equivalent in the form of additional plus for near vision, in patients *under forty*, because of lessened functioning power of the ciliary muscle, notwithstanding the frequent application of such lenses by medical practitioners of optometry. He has frequently had occasion to remove bifocals given by others, with results greatly appreciated by the patients.

When such necessity for additional plus or lessened minus existed, the case almost, if not quite, invariably had a history of atropine instillation into the conjunctival sac.

1. Optical Journal, February 12, 1914.

Furthermore, altho the writer has no actual figures on hand, he is aware that at least a high percentage of those who require *stronger* addition than their respective ages call for also have a history of atropinization.

In finality¹ he wishes to point out that his experience leads to the conclusion that the frequently atropinized eye becomes a hypersensitive eye, losing apparently not only muscular elasticity, but also neural elasticity, if the phrase may be used. In such cases, the patients, while possessing, as a rule, acute vision, are keenly and painfully aware of the slightest variation between their refractive error and the lenses worn. If the eyes change to the slightest extent or the lenses become altered from their accustomed adjustment, there is at once set up unbearable ocular pains and fatigue.

Then, too, there is the testimony of the patient who frequently says, "My eyes have never been the same since 'drops' were put in them." Even allowing for the fact that the patient frequently "does not know what he is talking about," this complaint is sufficiently common to merit consideration. Not seldom the sensation experienced by the sufferer greatly surpasses in accuracy the reasoning of science.

CONDITION OF BLOOD-PRESSURE. Owing to its power as a heart accelerator, the probable influence of atropine is to raise the blood-pressure temporarily, until the effect of the drug passes off. Hare² thinks recent investigations have thrown some doubt upon this theory, however.

This is all the information the writer has been able to gather from the medical literature at his command. If further information is extant, he has unfortunately been unable to discover it.

1. The subject of bifocals before the age of 40 is more fully dealt with in the *Keystone Magazine of Optometry*, October 8 and 22, 1914.

2. *Practical Therapeutics*.

CHAPTER 14.

SPHYGMOMANOMETRY IN OPTOMETRIC PRACTICE

The similarity between the ocular, cerebral and general symptoms of eye-strain, and the symptoms of those diseases which may or may not affect ocular health objectively, is so great at times as to render differentiation by the usual objective methods practically impossible. This is very evident when the etiology of such common symptoms as headache, nervousness, indigestion, nausea, vertigo, blurred vision, painful vision, muscular and retinal asthenopia, etc., is traced and found to originate from such widely divergent points as ocular strain, resulting from a slight refractive or muscular error, or from intestinal intoxication, renal, syphilitic or other diseases *apparently* confined to remote organs or areas.

The failure of the anomaly actually causing one or more of the mentioned symptoms to present objective ocular evidence undeniably pathognomonic of it is hardly conducive to confidence on the part of the operator or relief to the sufferer if the ocular evidence alone is relied upon. This confidence and relief are evidently in direct proportion to the skill and intelligence of the operator and to the extent of his knowledge concerning symptomatology.

The essential qualification, then, of any one engaged in optometric practice or, in fact, in any science, however

remotely related to therapeutics, is the ability to arrive at an accurate differential diagnosis.¹ Knowledge of symptoms other than local obviously becomes imperative, for it is becoming increasingly evident that the symptoms of ocular strain are simulated by the symptoms of many other abnormalities.

The use of sphygmomanometry for this purpose may be regarded as truly inspirational. The writer knows of no other science, nor can he imagine any, which covers such a wide field in pathology, is so happily applicable to ocular work, and combines with these qualities that of possessing such comparative simplicity. *Multum in parvo* is truly an appropriate phrase to be used in conjunction with it.

The great variety of diseases which effect blood-pressure changes, as indicated in the foregoing pages, and exert influences upon ocular economy or arouse symptoms which simulate those of refractive or muscular anomalies, has been partially enumerated. Their number, however, is of secondary importance in comparison with the frequency with which they intrude into optometric practice. A hundred diseases which are encountered in only five per cent of cases do not deserve the attention that may properly be directed to one which complicates ten per cent of cases. In other words, the value of knowledge concerning diseases and their symptomatology is in direct proportion to the percentage of optometric cases in which it may be practically applied.

In order to sustain his assertion that general diseases do enter into optometric practice in an amazing number of cases, and to give a comprehensive idea of this frequency, the writer presents herewith an analysis of two

1. In so far as this term can be applied to distinguishing between a state of health and a state of morbid disease.

hundred cases in which the blood-pressure was taken.¹ These two hundred cases are not selected ones, and may be taken as fairly representative of the general run:

Rx.	Sex	Age	S. P.	D. P.	P. P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
9	M	48	165			Reading causes strain.	Fundus pale. Arterial blood column irregular in intensity. (Physician established presence of kidney disease.)
10	F	52	132			Reading difficult.	Arteries slightly irregular in density. Slight exudate on nerve.
11	F	30	107			Pain on top of head.	Arteries—some normal, others pale. One unequal in caliber.
12	F	10	104			Eyes draw and ache.	Negative.
13	F	50	122			Local pain.	Negative.
14	F	11	95			Vision poor.	Negative.
15	F	51	128			Cephalalgia. Reading difficult. Local pain.	Negative.
16	F	34	114			Cephalalgia. R. epiphora. Nasal pain.	Negative.
17	M	28	132			Cephalalgia. "Blind spells."	Negative. (Specific disease?)
18	M	33	128			Eyes tire and ache. Epiphora.	Vein and nerve slightly thickened.
19	F	42	135			Distant vision blurs.	Negative.
20	M	54	130			Attacks of cephalalgia every 10 days. Reading wearies.	Fundus negative. (Physician says has tobacco heart.)
21	F	26	115			Epiphora.	Negative.
22	M	47					
23	M	18	112			Right eye weak. Left eye practically blind.	Negative.

1. It is assumed that the medical dictum concerning the pathology of cases outside the 100 mm. to 150 mm. limits is correct. The suggested influence in optometric work of cases with pressures between 100 mm. and 110 mm. is sufficiently well established in the writer's mind to be accepted as a fact, and these cases are, therefore, included in this consideration.

Rx.	Sex	Age	S.	P.	D.	P.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
24	M	54	127						Eyes hurt and blur.	Negative.
25	M	13	114	70	44				Frontal cephalalgia daily. Nausea.	Blepharitis. Conjunctivitis. Fundus negative.
26	F	7								
27	F	30	130						Right eye tires.	Negative.
28	F	18	105						Eyes tire and blur. Headaches, frontal and occipital.	Veins pale. Superior artery in R. E. disappears just before crossing vein.
29	M	51	165						Once a month or so has vertigo, nausea and emesis. R. E. draws. Indigestion.	Negative.
30	F	49	145						Distant vision blurs.	Negative.
31	M	44	118						Cephalalgia—occipital and frontal.	Vitreous opacities in fine dust-like form.
32	M	33	120						Vision blurs.	Negative.
33	M	81								
34	M	44	132						Frontal pains. Vision poor.	Negative.
35	M	27	136						Frontal cephalalgia.	Negative.
36	F	47	112						Reading difficult.	Negative.
38	F	52	170						Reading blurs and tires.	Negative.
39	M	50								
40	F	47	142						Nauseated. Cephalalgia. Tires easily.	Artery depresses vein. Apparent light streak along one artery.
41	M	37	112						Cephalalgia. Local strain.	Negative.
42	F	19								
43	F	12								
44	F	31	120	87	33				Cephalalgia. Emesis. Indigestion.	Negative.
45	M	32	137						Vision poor. "Cobwebs." Indigestion. Cephalalgia. Tires easily.	Negative.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
46	F	26	120					Vision blurs and tires.	Conjunctivitis. Fundus negative.
47	M	18	112 ¹ 118	80	35			Vertigo. Headaches weekly. Indigestion.	Negative.
48	F	32	130	94	36			Cephalalgia, frontal. Reading arouses nausea.	Negative.
49	F	45	116					Cephalalgia, frontal and occipital.	Negative.
50	M	74	156					Reading poor.	Cataracts.
51	F	44							
52	F	27	120					Sick headache.	Fundus negative. (Heart action irregular; admits has heart trouble.)
53	M	58	168 175	90	65			Vertigo. "Lenses focus too closely."	Nerve-head very dark gray, especially in center. (Facial coloring pasty white or yellow.)
54	M	30	155					Eye-strain. Eyes ache and tire. Vision poor. Car-sickness.	Arterial blood column irregular. Arteries depress veins and slight exudate on point of crossing. (Specific disease?)
55	F	56	140	85?	55			Reading and sewing difficult.	Negative.
58	M	37	125					?	Conjunctiva slightly inflamed.
59	F	48	120	80	40			Reading painful.	Negative.
60	F	34	110					Cephalalgia. Reading blurs.	Negative.
61	F	18	108					Vertigo. Cephalalgia. Eyes "sore."	Negative.
62	F	52	118					Reading blurred.	Negative.
63	F	55	130					Reading blurred.	Cataractous. Choroiditis. Patch of pigment on disk.

1. When a pressure is represented by two figures, they indicate a first and second time the pressure was taken:

Rx.	Sex.	Age	S.	P.	D.	P.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
64	F	27	118						Vision blurred.	Negative.
65	F	60	158						Vision blurred. Eyes tire.	Negative.
66	M	34	110						Cephalalgia. Local pain. Reading blurs.	Negative.
67	M	28	110						Occipital pains. Right conjunctivitis.	Negative.
68	F	22	116	80	36				Asthenopia. Cephalalgia.	Slight exudate (?) on nerve. Choroidal patch (small and uncertain).
69	F	54	126						?	Negative.
70	F	54	125						Reading difficult.	Negative.
71	M	51	138	88	50				R. E. pains. Large spot before R. E. Reading blurs.	Vitreous opacities. Fundus slightly hazy. Choroid atrophic near nerve. Pigment patch R. E. near nerve. (Specific disease?)
72	F	23	122 127						Cephalalgia, frontal and temporal.	Negative.
73	M	54	180						Old glasses not satisfactory.	Myopic fundus.
75	F	29	125	90	35				Cephalalgia, constant. Vision blurred.	Negative.
76	F	30	118	90	28				R. E. pains and tires.	Myopic fundus.
77	F		106	85	21				Cephalalgia. Indigestion.	Negative.
78	F	58	140						R. E. blind. L. E. partially. Vertigo.	Cataracts. (Heart action irregular.)
79	F	59	145	87	58				Temporal headache.	Arteries irregular in caliber and color. Slight aneurysm lower temporal artery. (Physician diagnosed nephritis.)
80	F	69	148	85	63				Reading and sewing blur.	Severe choroiditis.
81	F	5								
82	F	24	120	90	30				Asthenopia. Cephalalgia.	Negative.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
84	F	21	108	80	28			Cephalalgia.	Negative except slight conjunctivitis.
85	M	26	110					Cephalalgia. R. E. twitches.	Negative.
86	F	25	125	105	20			Cephalalgia.	Negative.
87	F	43	145 150	95	52			Cephalalgia. Indigestion. Eructation of gas. Reading blurs. Tinnitus.	Slight perivasculitis. Slight linear hemorrhage? Exudate on nerve-head.
88	M	28	120	85	35			Eyestrain. Black specks.	Negative.
89	F	32	95 100	80	18			Severe asthenopia. Blurred vision. Cephalalgia.	Retinitis. Two or three white and black spots on retina. Perivasculitis. Conjunctivitis.
90	F	56	140					Reading difficult.	Negative.
91	M	66	114	70	44			Wanted new glasses.	Negative. (Heart action very irregular.)
92	F	27	103	78	25			Cephalalgia. Pain in right eye.	Negative.
93	M	45	140	80	60			?	Apparent hemorrhage near disk.
94	F	15	107	65	42			Vision blurs.	Negative.
95	F	45	150					Reading blurs.	Patch of exudate near nerve R. E. Eruption of pigment near macula.
96	F	60	110	66	44			Nervous. Insomnia. Constipated. Indigestion.	Arcus senilis. Arteries depress veins. Superior vein distorted as artery passes over it.
97	M	33	110 115	80	32			Muscae volitantes. Close work bothers.	Arteries irregular in width and color. (Heart action irregular.)
98	M	22	124	65	59			Vision blurs.	Negative.
99	M	62	95 100	55 65	35			Eyes not comfortable. Nervous.	Vitreous opacities. Arteriosclerotic condition of vessels.
100	F	47	148 155	92 95	58			Local pains. Reading causes nervousness. Indigestion.	Arteries tortuous and uneven in caliber.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
101	M	52	124	78	46			Frontal pains. Reading blurs.	Arteries depress veins slightly in two places.
102	M	33	170	105	65			Nervousness. Indigestion. Vertigo. Objects distorted.	Left artery conceals vein. Vein concealed for 1 mm. by exudate. (Luetic disease?)
103	F	20	105	80	25			Cephalalgia. spells.	Blind Negative.
104	M	37	115	75 80	38			Nervousness. Indigestion.	Negative.
105	F	36	120	77	43			?	Inferior artery L. E. concealed for disk-width from nerve.
106	M	59	144	80	64			Reading difficult.	Only note is "Signs of retinal arteriosclerosis."
107	F	40	118	72	46			Cephalalgia. Eyes burn and smart.	Negative.
108	F	26	100 103	63 68	36			Asthenopia.	Negative.
109	M	48	118	80	38			Reading difficult.	Negative.
110	M	46	112					Cephalalgia. Indigestion.	Veins depressed by arteries.
111	F	11							
112	F	75	160?					Asthenopia.	Cataracts. Fundus otherwise negative.
113	F	22	110	80	30			Local pains.	Negative.
114	M	44	108	65	43			Tinnitus. Indigestion. Vertigo.	Slight exudate on nerve-head. Vein deflected by artery.
115	M	26	120 104	80 70	40 34			Nervous exhaustion.	Optic neuritis.
116	M	50	116	78	38			?	Negative.
117	M	55	152	105	47			Reading tires. Vision blurs. R. E. pains.	Retinitis. Arteriosclerosis.
118	F	43	112	90	22			Eye-strain. Drawing sensation.	Negative.
119	F	50	144 154	78	71			Vision blurs.	Slight optic neuritis. Luetic disease probably.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
120	F		130	88	42			Cephalalgia—frontal, temporal and occipital.	Retinitis pigmentosa.
121	M	35	118	70	48			?	Negative.
122	M	51	110	80	30			Epiphora. Eyes burn and smart.	Negative. Luetic disease probably.
123	F	48	112	80	32			Reading blurs.	Negative.
124	M	18	95	61	34			Eyes ache, photophobia. Headache, etc.	Negative.
125	F	16	120	80	40			Eye-strain, cephalalgia.	Negative.
126	F	41	95	65	30			R. epiphora.	Negative.
127	M	22	120	80	40			Vision blurs.	Negative.
128	F	45	260 270	150	115			Vision blurs. Occipital headache.	Pigment deposit. Macula mottled. Exudate. Vessels irregular in caliber. Typical picture of nephritis.
129	F	44	122	85	37			Asthenopia. Local pain.	Negative.
130	M	40	143 153	110	38			Vision blurs.	Negative.
131	M	24	100	73	27			?	Negative.
132	F	30	100 103	78 80	22			Cephalalgia. Local pain R. E. Vision poor.	Negative.
133	F	48	95	70	25			Cephalalgia. Reading tires.	Negative.
134	F	43	100 110	70	35			Photophobia.	Veins depressed by arteries. Latter bordered by exudate at point of crossing.
135	M	49	126	86	40			Reading bothers.	Negative.
136	F	45							
137	M	39	100	66	34			?	Disseminated choroiditis.
138	F	21	132					Cephalalgia, frontal and occipital.	Negative.
139	M	40	120	76	44			Asthenopia. R. E. becomes hyperemic after use in reading.	Negative.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
140	M	47	94 98	68	32			Tinnitus. Reading blurs.	Negative.
141	M	21	98	70	28			?	Negative.
142	F	59	135	90	45			Muscae volitantes.	Negative.
143	M	77							
144	F	54	195	120	75			Nervousness. Vision poor.	Veins markedly depressed. Deflected by arteries.
145	F	53	164 170	90 100	72			Cephalalgia. Indigestion. Blurred vision.	Vertigo. Negative.
146	F	37							
147	M	25	125	87	38			Vision blurs. Local strain. Cephalalgia.	Neuritis. Pigment deposits.
148	M	58	130	90	40			Reading blurs.	Choroidal vessels pronounced and light in color. Sclerosed?
149	F	24	105 100					Cephalalgia.	Negative.
150	M	25	102	69	33			Occasional headache.	Negative.
151	M	51	106	83	23			Reading difficult.	Deposit white exudate R. nerve-head. Arteries show broad light streak.
152	M	35	136	100	36			Vision blurs.	Slight exudate in L. E. near nerve and over vessel.
153	M		116 120	74	44			?	Negative.
154	F		103					Asthenopia.	Negative.
155	M	27							
156	M	53	151 160	90 95	61 65			Cephalalgia. Drowsiness.	Mottled appearance near macula. Phlebosclerosis.
157	F	14	112	80	32			Vision poor.	Optic nerve degeneration. (Heart irregular. Luetic disease probably)
158	M	51	136	60	76			Pain in neck, spine and shoulders. Indigestion. Nervousness.	Slight optic neuritis. Vascular signs of arteriosclerosis.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
159	M	14	120 105	65	47			Vision poor.	Negative. (Luetic disease?)
160	M	29	125	78	47			Nausea. Pain in forehead and occiput.	Left conjunctivitis.
161	M	47	95	70?	25			Strain in reading. Blur.	Slight exudate on veins on disk.
162	M	78							
163	M	32	132	90	42			Asthenopia.	Arteries depress veins.
164	F	57	130 134	75 80	54			Vision blurs.	Negative.
165	F	25	132					Asthenopia. Cephalalgia. Vertigo.	Negative. (Doctor says has auto-intoxication.)
166	F	49	170					Reading difficult.	Retinitis. Optic neuritis.
167	F	56							
168	F	41	105	77	28			Occipital pain. Eye-strain.	Negative.
169	M	35	135	92	43			Cephalalgia, temporal and occipital.	Inferior temporal quadrant of nerve atrophic. (Heart trouble.)
170	F	49	135	80	55			Cephalalgia, temporal and occipital. Vision poor.	Slight edema.
171	F	52	130?	80	55			Vision blurred. Eyes ache.	Diabetic retinitis.
172	M	46	130	80	50			Cephalalgia. Nervousness. Blurred vision.	Temporal half of each nerve pale. Arteries tortuous.
173	M	50							
174	F	57							
175	F		110					Vertigo. Cephalalgia. Eyes pain.	Negative.
176	F	38	110 115	78 80	35			Nausea. Cephalalgia, frontal and occipital.	Negative.
177	M	24							
178	F	26	100	70	30			Reading blurs.	Arteries irregular in color, width and form.

Rx.	Sex	Age	S.	P.	D.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
179	F	24	120	80	40			Eyes blur and tire.	Negative. (Heart action irregular.)
180	M	31	100	78	27			Vision blurred.	Negative.
181	M	46	175 155	98 95	77 60			Vertigo. Cephalalgia.	Retinitis. Optic neuritis.
182	F	50	110	75	35			Vision blurred.	Retinitis?
183	F	36	110	78	32			Cephalalgia. Vertigo. Nausea.	Negative.
184	F		101 102					Cephalalgia.	Negative.
185	M	19	115 118	73 75	43			Cephalalgia. Emesis.	Negative.
186	M	20	105	70	35			?	Fundus hyperemic?
187	F	24	92 105	65 65	27 40			Cephalalgia.	Pupils very small.
188	M	46	110 115	68 70	45			Vertigo. Reading blurred.	Negative.
189	F	47	125	87	38			Headache. Reading blurs.	Arteries irregular and hidden in places as tho by exudate.
190	M	48							
191	M	71							
192	F	75	150+					Asthenopia and epiphora. Vision blurs.	Cataract.
193	F		120	65	55			Cephalalgia, frontal and occipital. Asthenopia.	Negative.
194	F	71							
195	M	77	170	85	85			Vision blurs.	Choroiditis.
196	M	49	115	75	40			Cephalalgia.	Negative.
197	M	48	155	95	60			Cephalalgia, frontal, temporal and occipital.	Faint perivasculitis.
198	F	80							
199	F	48	158	92	66			Cephalalgia. Indigestion.	Perivasculitis? Coloring of optic nerve irregular.

Rx.	Sex	Age	S.	P.	D.	P.	P.	P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
200	M	45	118	80	38				Reading difficult.	Negative.
201	F	41	117	80	37				Vision poor. Temporal pains.	Negative.
202	F	63	120						Cephalalgia.	Negative.
203	F	81	120?						Frontal pains. Eyes itch.	Negative.
204	M	76								
205	F	54	155	80	75				Vision blurs. Vertigo.	Negative.
206	M		112	68	44				Indigestion.	Negative.
207	M	60	105	62	43				Vision blurs.	Negative.
208	M	33	112	70	42				Asthenopia.	Negative.
209	F	69	110						Vision blurs.	Negative.
210	F	49	112	75	37				Cephalalgia. Vision poor.	Negative.
211	M	39	110	80	30				L. E. feels strained.	Inferior artery tortuous and partially hidden.
212	F	25	105	75	30				Cephalalgia. Asthenopia severe.	Negative. (Hutchinson teeth.)
213	M	65	98	62	36				Reading blurs.	Negative.
214	M	40	122	84	38				Cephalalgia.	Negative.
215	M	72								
217	F	49	170 175	95 100	75				Cephalalgia. Indigestion. Vision blurred.	Choroidal disturbance nasal side R. nerve.
218	F	40	170 175	100 105	70				Reading blurs.	Negative.
219	M	29	100	75	25				Cephalalgia. Asthenopia.	Negative.
220	F	45	180						Ocular pains.	Negative.
221	M	18	128	88	40				Eyes smart and burn.	Negative.
222	M	63	218	105	113				Cephalalgia. Transient blindness.	No marked signs of ocular abnormalities.
223	M	40	112	70	42					Negative.
224	F	57	205	110	95				Cephalalgia. Indigestion. Vision blurs.	Arteries pale, rigid; deflect veins.

Rx.	Sex	Age	S.P.	D.P.	P.P.	Subjective Symptoms	Objective Symptoms (Chiefly Ocular)
225	F	65	156			Vision blurs.	Choroiditis.
226	F	14	105	80	25	Cephalalgia. Vertigo.	Negative.
227	M	23	105	70	35	Sick headache. Vertigo.	Negative.
228	F	47	153 145	75 72	78 73	Reading blurs.	Negative.
229	F	45	126	85	41	Cephalalgia. Vertigo. "Half vision" at times.	Negative.
230	M	56	142	82	60	Asthenopia. Cephalalgia.	Negative.
231	M	77					
232	F	12	93			?	Negative.
233	F	48	115?	84	31	Vision poor.	Negative (except one pupil is smaller than other).
234	F	79	190			Vision poor. Vertigo.	Cataract. Vitreous opacities.
235	M	28	115	67	48	Indigestion. Nausea. Cephalalgia.	Negative.
236	M	26	130	60	70	Cephalalgia, frontal and occipital. Indigestion.	Infero-temporal quadrant of L. nerve gray. Otherwise negative.
237	F	65	160	78	82	Vision blurs.	Vein decreases in caliber as approaches disk.
238	M	33	126	78	48	Asthenopia.	Negative.
240	F	59	240			Vertigo. Frontal pains. Vision blurred.	Prominent signs of arteriosclerosis.

Fig. 19
Table of Author's 200 Cases

Careful scrutiny of this number reveals the information that thirty-six, or eighteen per cent of them, possess a blood-pressure of 150 mm. or over; twenty-eight, or fourteen per cent, have a blood-pressure between 100 mm. and 110 mm.; nine, or 4.5 per cent, a pressure under 100 mm. Of thirty, out of 125 cases in which diastolic

pressure was taken, who have an abnormally high pulse pressure, there are twelve, or 9.6 per cent of 125, not included in the above figures (*i.e.*, there are twelve whose systolic pressure appears normal) and two, or 1.6 per cent, not included whose pulse pressure is too low.

COMPARATIVE ANALYSIS

It is interesting to note what proportion of these cases displaying an abnormal blood-pressure reading also yield ophthalmoscopic evidence of pathologic conditions. The following table presents this information, giving, also, under each amount its percentage of the entire 200 cases:

	Cases with pressures relatively normal	Cases with pressures of 150 mm. or more	Cases with pressures between 100 & 110 mm.	Cases with pressures under 100 mm.	Total
	127	36	28	9	73
Absence of pathologic fundus conditions found in.	93 = 73% 1. 46.5%	10 = 28% 5%	17 = 60% 8.5%	7 = 77% 3.5%	34 = 46% 17%
Doubtful evidence of pathologic fundus conditions found in.....	13 = 10% 6.5%	9 = 25% 4.5%	6 = 21% 3%	1 = 11% .5%	16 = 22% 8%
Positive evidence of pathologic fundus conditions found in.....	21 = 16% 10.5%	17 = 47% 8.5%	5 = 18% 2.5%	1 = 11% .5%	23 = 32% 11.5%

Fig. 20

Analytical Table No. 1.

1. These lower percentages refer to the relationships the accompanying number of cases (93, in this instance) bears to the total number of cases (200).

Summarized, this table in its application to the entire 200 cases is presented as follows:

	Percentage of cases in which each instrument yielded evidence	Percentage of cases in which both instruments yielded evidence	Percentage of cases in which only one instrument yielded evidence
Ophthalmoscopic evidence (positive and doubtful).	36.5%	19.5%	17%
Sphygmomanometric evidence (positive)	36.5%		17%

Fig. 21

Analytical Table No. 2.

Strangely enough, the summarized table presents the relative value of the ophthalmoscope and sphygmomanometer as exactly the same. However, there are not included in the figures representing the latter instrument those relating to abnormal pulse pressure.¹ These constitute a doubtful element, but one certainly not more doubtful than many, or all, of the cases presenting what is termed "doubtful" ophthalmoscopic evidence of pathologic conditions.

Among the latter are probably many in which the abnormalities noted were merely physiological departures from the normal. The variations of a healthy fundus are so numerous that it is difficult to tell, at times, where health leaves off and disease begins. Nevertheless, whenever doubt existed, it was so noted, that a fair analysis might be made.

1. Special reference is here made to those cases possessing an abnormal pulse pressure, but a relatively normal systolic pressure.

To be equally fair, cases presenting an abnormal pulse pressure should be included in the table. They certainly indicate, according to medical authorities, abnormal conditions generally, tho the writer is not yet prepared to say what influence such cases have upon the eye.

If such cases are included in a similar table, and if the fourteen cases discovered in the 125 cases in which the diastolic pressure was taken may be assumed to represent the general average, the following tables more truly represent the relative values of the two instruments:

	Cases with pressures relatively normal	Cases with pressures of 150mm or more	Cases with pressures between 100 & 110 mm.	Cases with pressures under 100 mm.	Cases with abnormal pulse pressure	Total
	105	36	28	9	22	95
Absence of pathologic fundus conditions found in...	82=78% 41%	10=28% 5%	17=60% 8.5%	7=77% 3.5%	11=50% 5.5%	45=47% 22.5%
Doubtful evidence of pathologic fundus conditions found in...	10=9% 5%	9=25% 4.5%	6=21% 3%	1=11% .5%	13=3% 1.5%	19=20% 9.5%
Positive evidence of pathologic fundus conditions found in...	13=12% 6.5%	17=47% 8.5%	5=81% 2.5%	1=11% .5%	8=36% 4%	31=32% 15.5%

Fig. 22

Analytical Table No. 3

Summarized table, in its application to 200 cases:

	Percentage of cases in which each instrument yielded evidence	Percentage of cases in which both instruments yielded evidence	Percentage of cases in which only one instrument yielded evidence
Ophthalmoscopic evidence (positive and doubtful)....	36.5%	25%	11.5%
Sphygmomanometric evidence (positive and doubtful)....	47.5%		22.5%

Fig. 23
Analytical Table No. 4

Included in none of these tables are those cases in which third-phase weakness exists, altho the writer is morally certain such cases are of optometric interest; nor are there included other cases displaying symptoms of abnormalities of doubtful optometric interest, such as cardiac irregularity, etc., unless such cases are incidentally included because of the other more pertinent symptoms.

Altogether, no ill-advised effort is made to inflate the figures representing the comparative value of the sphygmomanometer in optometric work. On the contrary, the writer feels that his presentation of this value is essentially conservative. There is no doubt in his mind concerning the existence of several phases of blood-pressure readings not yet established as of optometric concern, but which, nevertheless, do merit such consideration.

It seems obvious that the conclusion to be drawn from this analysis is that the sphygmomanometer is at least equal in value to the ophthalmoscope in optometric practice.

Eliminating those cases between 100 mm. and 110 mm., and also those having an abnormal pulse pressure, there is a total of 22.5 per cent of cases in which there is

practically undeniable evidence of pathologic conditions other than ocular. *Including* such cases—and the writer thinks it is no more than fair to include them, in view of his experience with them—*there is a grand total of 47.5 per cent of optometric cases in which sphygmomanometric evidence of abnormal conditions may be found.*

On the other hand, in only 36.5 per cent of cases was ophthalmoscopic evidence, positive and doubtful, found, while in only 22 per cent was positive evidence alone discovered. It should be remembered, too, as elsewhere suggested, that while the latter evidence is termed “positive,” radical anomalies may exist upon the fundus of the eye and have no connection or association with pathologic conditions elsewhere, being merely congenital freaks of Nature. Such cases are comparable, perhaps, to those cases in which abnormal blood-pressure exists without there being any assignable cause. They are particularly analogous to congenital cardiac anomalies.

This is less true of radical departures from normal of the blood-pressure. High blood-pressure, if sustained, is practically certain to ultimately terminate in a degeneration of the vessel walls or in the destruction of ocular tissue. Whether the pressure proper is the cause of such destruction, or whether it is secondary to other pathologic conditions, is immaterial under present considerations. Low pressure is a factor of equal importance in optometric work, tho for different reasons, as later comments will indicate.

Even modifying to ten per cent the proportion of optometric cases in which information of value is obtained by the sphygmomanometer, there is sufficient justification for the assertion that the instrument should be used in every case. It is probable that this proportion,

absurdly low as it is under the circumstances, is much higher than that applying to many instruments, or methods, utilized by the medical profession and considered indispensable by it.

On the whole, this analysis constitutes a somewhat startling revelation, and one the writer has hesitated to promulgate for fear of being thought an extremist, but careful thought has resulted in the conclusion that these figures do not in any way misrepresent facts. Even tho some of them may undergo modification as a much greater amount of material is gathered, it is as true that others may be as greatly augmented and that the net result will therefore remain practically the same.

The average systolic pressure of the two hundred cases has been found to be 127.2 mm.; the average diastolic 79.2 mm., and the average pulse pressure 47.4 mm. For men alone, the average systolic pressure is 125.4 mm.; for women 128.7 mm. For men whose pressures ranged from 100 mm. to 150 mm., the average was 120 mm.; for women 120.2 mm.

These figures differ to some extent from those of writers who have made similar investigations, but these differences are not necessarily of great consequence, and may be attributed to the fact that included in the two hundred cases are many cases of disease. Other investigators have apparently confined themselves to healthy individuals.

The average systolic pressure of 127.2 mm. is slightly high when it is considered that women are included in the count, and this may properly be attributed to the fact that several very high pressures are registered. The average diastolic pressure of 79.2 mm. is rather low, a circumstance which may or may not be accepted as characteristic of the class of individuals who are susceptible to eye strain. The writer is inclined to accept this in the affirmative. The average systolic pressure of men alone is rather low, a fact which merits a suggestion similar to the one just mentioned—the susceptibility of low-pressure individuals to the symptoms of ocular strain.

On the other hand, the average systolic pressure of women—128.7 mm.—is unusually high, being elevated above that generally found by several very high pressures among the two hundred cases. Where there are only fourteen men with a pressure of 150 mm. or more, there are twenty-two women, a condition which evidently does not usually exist and which may be taken as exceptional.

The average systolic pressure of men in whom the pressure ranges from 100 to 150 mm. is somewhat low (120 mm.), probably for the above-mentioned reason. Warfield finds the pressure under somewhat similar conditions to be about 127 mm. For women, the 120 mm., is exactly as Warfield found.

It should be borne in mind also that many cases are fit subjects for medical attention who, nevertheless, display nothing abnormal in either of the three pressures. Such, for example, are those in which the third phase is sufficiently weak to indicate cardiac trouble, or those in which an abnormal relationship between the three pressures has existed, but has been replaced by a temporary normal one incidental to an imminent collapse. The latter condition is quite possible when a weakening heart has resulted in changing the pressures from 180 mm. systolic, 100 mm. diastolic and 80 mm. pulse pressure to 130 mm. systolic, 100 mm. diastolic and 30 mm. pulse pressure, a change not at all impossible in cases of arteriosclerosis in which the heart can no longer sustain the pressure necessary to adequate peripheral circulation.

That the study of these cases may be simplified, it has been found advisable to divide them into groups.

GROUP A consists of those cases in which a systolic pressure of less than 100 mm. is registered.

GROUP B, of those cases in which a systolic pressure from 100 mm. to 110 mm. is registered.

GROUP C, of those from 110 mm. to 150 mm.

GROUP D, of those from 150 mm. to 170 mm.

GROUP E, of those from 170 mm. up.

GROUP A

In studying groups, it is again found that division is necessary; hence the following classifications:

CLASS 1. Included in this class are those cases having a systolic pressure under 100 mm., but which do not present any other symptoms of abnormalities sufficiently great to command attention.

For example, Case 141 may be cited. This was a young man of about twenty-five years, in apparently good health and with very few symptoms of ocular strain. His refractive defect is represented by the following formula:

O. D. + .62 + .12 axis 65 V. A. 15-13

O. S. + .25 + .50 axis 105 V. A. 15-13

Esophoria one and one-half degrees.

Lenses made accordingly gave satisfaction and, since the three pressures were in approximately correct proportion to one another, the case may properly be regarded as one in which the hypotension is but temporary and well compensated, or normal to that particular individual (the man was very undersized, which may have had something to do with the pressure, tho most authorities do not believe size has a perceptible influence). In view of the fact that there was no evidence of pathologic ocular changes, that lenses gave relief from the symptoms complained of and that there was no other evidence of general disease, the case may properly be discharged in so far as the general condition was concerned, and no responsibility in that respect accepted.

This policy should apply to practically all children under fifteen years, of course, but only to a very small proportion of cases over that age—cases in which glasses give complete relief and in which there is no evidence, either objective or subjective, of other abnormalities.

CLASS 2. This class may be said to include those cases in which glasses supply the patient's wants, but in which there is unmistakable evidence of abnormalities other than ocular.

Case 213 typifies such a patient. The man, aged sixty-five and appearing even older, wanted glasses solely for reading. Lenses supplied for this purpose gave complete satisfaction, but there was such conclusive evidence of other abnormalities that it was thought advisable to suggest the desirability of having a general physical examination made. The operator must be governed, in such cases, by discretion, for where one patient may take kindly to such a suggestion, another may deeply resent it and suspect ulterior motives. Especially is this true if the patient has not already experienced symptoms of approaching collapse.

CLASS 3. Patients in this class consist of those who possess appreciable refractive or muscular errors and undeniable symptoms of eye-strain, but who fail to obtain full or perhaps even partial relief from the symptoms by the correction of the defects.

Case 99 well illustrates this type. The patient, a man of sixty-two, nervous, irritable and dyspeptic, suddenly developed approximately half a diopter of hyperopia, after having, for many years, about one-half a diopter of simple hyperopic astigmatism which still remained. The correction of this ametropia failed to

give relief and the complaint of blurred vision was persisted in, even tho the plus element was finally reduced until there was absolutely no question of the lenses being too strong. Annoying ocular discomfort was complained of in spite of everything that the writer could do.

Objectively there existed vitreous opacities, well-defined evidences of sclerosis of the retinal arteries, and tortuous temporal arteries. As can be observed, the sphygmomanometer revealed a systolic pressure of 95 mm. and a diastolic of about 60 mm. There was unmistakable evidence of cardiac anomaly.

The patient was sent to his physician, who made an examination(?) and reported that there was nothing wrong with him! Under the circumstances there was nothing to do but accept the verdict, which the writer did—with mental reservations.

Case 89, while listed under cases having a pressure between 100 to 110 mm., may be cited as an example in this class, as subsequent examinations gave a systolic pressure of less than 100 mm.

This patient, a nurse of thirty-two years, came to the writer with a complaint of headache, blurred vision, photophobia, etc. An examination revealed refractive and muscular defects as follows:

O. D. — .25 — .25 axis 175 V. A. 15-13

O. S. — .75 — .62 axis 145 V. A. 15-13 partly

Hyperphoria one degree. Exophoria twelve degrees.

She was wearing glasses upon the following formula:

O. D. — .25 — .25 axis 165

O. S. — .87 — .62 axis 135, $2\frac{1}{2}^{\circ}$ base down.

Her lids were red and inflamed and retinitis was present to a marked degree. There was also one small white and one small black spot located upon the fundus.

Not wishing to jump to conclusions too hastily when the low blood-pressure was discovered (all sounds were weak with the stethoscope, a symptom of cardiac weakness), and realizing that much of her trouble could arise from the improper lenses she had been wearing, and her uncorrected muscle imbalance, the writer gave her lenses which included a three-degree prism base in each eye.

Her report in two weeks was that while part of her symptoms had disappeared, there still remained considerable discomfort. Not finding that her lenses could be changed to advantage, she was instructed to visit a physician and have a general examination made. This was done, and he reported a state of auto-intoxication which required an operation.

The operation was performed and the patient sent home to recuperate. Upon the elapse of several weeks, she returned and reported that tho there was further improvement, she still had ocular discomfort, photophobia existing to a marked degree.

A careful refraction and muscle examination revealed practically nothing other than that which had been previously discovered, so nothing further could be done except give lenses which would tend to ameliorate the photophobia by reducing the illumination. This was accomplished by means of Euphos glass, and the effect was very pleasing.

However, there was still remaining more or less ocular discomfort, especially when reading was attempted, and the patient was assured, by both the writer and the physician, that nothing further could be done at present, and that she now needed a complete rest that full recovery from the effects of the operation might be accomplished. At any rate, the writer is confident that

all has been done ocularly that can be done, and that if further attention is necessary it must be medical.¹

Case 124 presented practically the same symptoms as the preceding case. The patient, a young man of eighteen, gave a history of dull eye-ache, blurred near vision (spasmodic), severe photophobia, occipital headache. The lids were highly inflamed.

He was wearing

O. D. + .25 + .50 axis 90 1° base down

O. S. + .25 + .50 axis 90

Add + .75 for reading.

These lenses were prescribed by a very excellent medical refractionist.

The writer found necessary

O. D. + .50 + .75 axis 90 2° apex in at 170.

O. S. + .50 + .50 axis 80.

There were three degrees of esophoria at the angle indicated, and with these lenses perfectly normal vision was obtained. They were given in light shade Euphos glass.

As the action of the muscles was very sluggish, muscle calisthenics were advised and given, three weekly for one month. The effect at first was distinctly uncomfortable, pain and profuse epiphora resulting.

Later, as the exercises continued, this subsided to some extent. It was also noted that after the internal rectus had been stimulated several times at one sitting by prisms bases out, binocular vision became increasingly difficult, showing, apparently, a reluctance or inability of the external rectus to rotate the eye back to a state of parallelism. It is not likely that the condition was due

1. The patient has since reported that a rest of two or three months has resulted in complete relief.

to resistance offered by the internal rectus muscle, and there is a note to that effect on the record, there also being a suggestion that the condition was due to an inflammation at the base of the brain, indicating the writer's belief at that time that the condition was due to a luetic congestion.

The lenses and exercises given resulted in a modification of the symptoms complained of, but their complete abatement did not occur, and, as a blood-pressure of 95 mm. systolic and 60-62 mm. diastolic was discovered, the family physician was communicated with. He proved non-committal, stating that he thought "there was something wrong of which the writer was not aware." He made no comment about the blood-pressure, which was undeniably low, even for so young a man.

After a month or two, the patient resumed his school work and the symptoms originally complained of again increased in severity. Additional plus for reading alone had no perceptible effect. Close application resulted in severe ocular pains, and his lids were swollen and inflamed markedly.

Finally, the patient called again to see if further relief could be obtained. The eyes were gone over most carefully, and positively not the least change in the glasses being worn found permissible. The blood-pressure was also taken and found to be about the same as before.

In desperation, his physician was again called up and the case stated explicitly to him. In reply he said that altho he had made a Wassermann test and had been unable to demonstrate the presence of congenital luetic disease, he was morally sure the patient suffered from that condition as he not only presented many of the subjective

and objective symptoms of the disease, but also that his parents had both suffered from it, due to an earlier infection of the father.

Here was sufficient confirmation of the writer's suspicion as recorded two months previously, and so the patient was advised that nothing further could be done ocularly and that general medical attention was required in order to achieve results.

In this case there were several symptoms of a condition other than the local functional strain, but none of them were quite as pronounced as the blood-pressure reading, unless the condition of the extrinsic muscles could be so considered. Even the latter was of questionable importance unless considered in conjunction with other symptoms. Similar muscular disturbances can result without the cause being deep-seated or other than merely temporary.

So the final differentiation depended actually upon the *combination* of local symptoms with a symptom hitherto neglected—the blood-pressure reading. If practically similar circumstances exist in future cases, the associated phenomena can be looked upon as more or less pathognomonic of the disease, especially as the abducens nerve leads in the order of frequency of ocular muscle anomalies, and, as one writer quoted asserts, such muscle anomalies in themselves alone may be often regarded as pathognomonic of luetic disease.

There are many such cases, and they are usually the despair of the refractionist; they vainly try to obtain ocular relief by going the rounds of both oculists and optometrists, when medical attention to remote disorders is what is required. They possess most pronounced

symptoms of ocular strain and are very persistent in their attempts to obtain relief. They are usually found, however, in the next group.

GROUP B

In this group there are likewise three classes—those who have symptoms of ocular strain, which lenses relieve, but no other marked evidence of abnormalities susceptible of revelation by optometric methods; those in whom there are symptoms of ocular strain which lenses relieve, but who *do* have symptoms of other abnormalities, but are not necessarily aware of them; and lastly, those who have symptoms of ocular strain and sufficient ocular anomalies to cause them, but who obtain little or no relief by wearing lenses.

CLASS 1. This class differs not very greatly from Class 1, Group A. It can be mentioned, however, that included in it are many cases in which remarkable relief is obtained by the wearing of weak lenses which correct either a slight hyperopia, a slight astigmatism or a slight muscle anomaly (chiefly esophoric), or any combination of them.

Case 77, for example, serves as an illustration. This patient, a young married woman of about twenty-five years, complained of most distressing headaches and digestive disturbances which had persisted for some time.

She was wearing + .50 D. S., O. U. A refractive and muscle examination revealed the following condition:

O. D. + .25 + .25 axis 85 V. A. 15/13

O. S. + .12 + .37 axis 100 V. A. 15/13

Esophoria three degrees.

Lenses upon this formula were given, together with a one and one-half degree prism base out, left eye, and the

patient instructed to return in a few weeks. This she did and reported her delight with the new glasses and their effect in *totally* relieving her trouble.

In spite of the relatively low blood-pressure in this case (the pulse pressure is particularly low, being only 21 mm.) and the fact that there *apparently* exists at one point of the fundus of one eye a linear extravasation of blood along one vessel for a distance of one or two millimeters, the writer did not feel justified in urging medical attendance, since questioning elicited the information that she had been treating with a physician for some weeks for the condition of which she complained when in the writer's office—without result. Inasmuch as the lenses given completely relieved her of the conditions complained of, and as she had just received the attention of her family doctor, the writer felt his obligations fulfilled. Responsibility for further developments rests with the attending physician.

CLASS 2. This class could not be better illustrated than by Case 151.

This patient, a banker of fifty-one, who has been coming to the writer for years for reading glasses, presented himself for an examination with the statement that he thought his reading was not accomplished with the comfort which should exist.

A refraction revealed the necessity of half a diopter addition to the lenses which he was already wearing, a circumstance to be expected since the last lenses had been given about two years previously.

Upon examining with the ophthalmoscope, it was noted that there existed in the right eye, adjacent to the lower side of the nerve, a relatively large white patch,

about half the size of the nerve-head; no previous records mentioned the existence of this patch, so it was naturally assumed to have formed since the last examination.

The arteries also showed signs of arteriosclerosis, having the pale appearance and broad light streak so characteristic of this condition. The heart action was very irregular, as indicated by the movement of the hand on the dial of the sphygmomanometer, and, as stated, the blood-pressure was systolic 108 mm., diastolic 82-84 mm. and the pulse pressure 24-26 mm.

The patient was advised to go to his physician and have an examination made, particularly of his kidneys, altho he made no complaint regarding his general condition and, upon questioning, stated that he felt nothing wrong except that he was very tired of late, a condition he expected to remedy by starting a two weeks vacation, within a few days.

However, he went; the physician made an examination and reported that the vacation was all the patient needed. He ridiculed the idea of the patient's having kidney trouble, and so the patient went home thoroughly imbued with the idea that the previous examiner "did not know what he was talking about." (It should be mentioned, *en passant*, that the writer did not then, and never does, assert that the patient had kidney disease. He merely suggested that an examination might be advisable and that the physician's word might better be accepted as the opinion of one skilled in such work in which the writer professed no skill whatsoever.)

Two days later, the patient collapsed in his office, was rushed to the hospital by his physician who had the intention of operating for appendicitis, and there dis-

covered to have the very condition the writer suggested—kidney disease. The operation was not performed.

Such cases as this—and they are not infrequent—naturally arouse justifiable doubt concerning the thoroughness and adequacy of many physicians' examinations. All authorities seem to agree that some conditions, kidney disease for example, cannot always be diagnosed by one urinalysis, and that very often the blood-pressure is much more reliable than the urinalysis, yet many times the writer has sent patients to physicians with the request that they examine for this disease, giving the physician the blood-pressure, which might or might not be markedly abnormal, and the intraocular condition, which also might be markedly abnormal, only to have him make one perfunctory urinalysis and completely dismiss the patient with the assertion that he or she was "all right." This slipshod method of totally ignoring an abnormal blood-pressure or the abnormalities of an organ—the eye—of the greatest importance in the symptomatology of many diseases—abnormalities, which are often pathognomonic of certain diseases—certainly is reprehensible and should be rectified.

As long as such methods do exist, an optometrist who sends to a physician a case having very evident symptoms of abnormalities other than ocular, only to have the report sent back that "nothing is wrong," need not feel discouraged and jump to the conclusion that his own observations are not correct. Perhaps they are not, but, on the other hand, it is just as likely that the other fellow is wrong.

CLASS 3. This class is likewise similar to Class 3, Group A. Characteristic of it is Case 212. This patient, a young married woman of twenty-five, complained of

ocular pain, headache, dry feeling of the eyes and weariness after reading.

She had had the lenses she was wearing for about six weeks, when her call at the writer's office was made. They were + .25 cyl. ax. 90 O. U. Her lids were red and irritated, more or less photophobia was present and the fundus rather hyperemic. A refraction revealed:

O. D. + .37 + .12 axis 90 V. A. 15/13

O. S. + .25 + .25 axis 100 V. A. 15/13

The sphygmomanometer gave the following pressures: systolic 105 mm., diastolic 75 mm., pulse 30 mm.

This woman had the well defined Hutchinson teeth described on page 156, and this fact, combined with the above-described ocular and sphygmomanometric findings, led to a very guarded prognosis. Crookes glass, upon the formula mentioned, was given, and the result was, as expected, partial but not complete relief.

It is very evident that in this case there is toxemia in one form or another and that too great assurance of relief of the symptoms complained of by the wearing of lenses is not at all justifiable. Whether or not this toxic condition is the result of congenital luetic disease cannot be determined by the writer, as it is a matter for the physician. The patient's dentist had suggested to her the possibility of such a circumstance and advised the Wassermann test, but when she was last seen this advice had not been followed. It is obvious that ordinary circumstances would not justify an optometrist in making such a suggestion.

A somewhat similar condition existed in Case 184. The patient, also a young married woman of less than thirty, complained of exhausting headaches and ocu-

lar signs of weakness. Her refraction was 1 D. O. U. hyperopic. Lenses failed to give entire relief. It was impossible in this case to hear any phase of the blood-pressure except the first, and this was exceedingly weak, being barely discernible. If the action of the dial-hand were transferred to a revolving drum, its registration would take the form of the wave-like line illustrated at *b*, Fig. 24, instead of the normal sharp, peak-like line at *a*. The latter is indicative of a strong, vigorous heart, while the former indicates the converse. Cardiac weakness was, therefore, very apparent, and the patient was advised to continue treatments which her physician was giving.

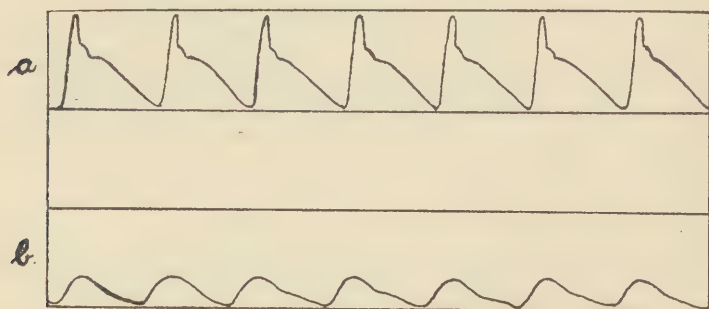


Fig. 24

a , Normal pulse; *b*, tardus pulse.

It is because of the comparative frequency of such cases among those having a systolic pressure of 100 to 110 mm. that it has been deemed advisable in optometric work to raise the lower limits of the normal elasticity of the systolic pressure to 110 mm. instead of permitting it to remain at 100 mm., the point at which pathologists in general seem to think it should be placed. Continued experience tends to increase, rather than modify, this belief.

GROUP C

This group is subject to a classification similar to the previous ones, but with the exception that a far higher percentage of cases comes under Class 1 than in the other groups. Greater confidence that lenses correcting an appreciable ocular anomaly will give relief from the ordinary symptoms complained of is justifiable.

Nevertheless, there do occur cases similar to those in Groups A and B, Class 3¹, and they must be carefully sifted from the rest.

CLASS 3. Case 158 is an example of one type. This patient, a man of fifty-one, stated that he suffered from indigestion, nervousness, pain in neck and spine and under shoulder blades, and blurred vision in reading.

He was wearing:

O. D. — .50 — .75 axis 160

O. S. — .75 — .75 axis 80

Add + 1.50 for reading.

These lenses were in the flat Kryptok form. The writer's refraction indicated the defect to be:

O. D. — .50 — .75 axis 150 V. A. 15-13

O. S. — 1. — .50 axis 85 V. A. 15-13

Add + 2. for reading.

These were made up in toric Kryptoks, light shade Crookes glass.

It will be observed that the difference between the two prescriptions is slight and could hardly account for all the symptoms the patient had been having. The ophthalmoscope revealed a slightly hyperemic disk, pale arteries, and veins depressed and bent in the direc-

1. Mention of Class 2 is omitted as it is identical with Class 2 in Group B.

tion of the arterial flow at the points where arteries crossed them—the latter being unmistakable signs of arteriosclerosis.

The blood-pressure was found to be systolic 136 mm., diastolic 58-62 mm., pulse 74-76 mm. The patient stated that six weeks previously his physician had found a systolic pressure of 180 to 200 mm., which he says has existed for years. Later this dropped to 120 mm. and then to 112 mm. At the latter point, the patient was sent by the physician to a health resort for treatment, and he had returned to the city only a few days before his visit to the writer. His trip and treatments had had no *apparent* effect inasmuch as he felt no better, altho the rise in blood-pressure from 112 mm. to 136 mm., could, perhaps, be viewed as encouraging.

Notwithstanding this, it was very evident that the patient was in a serious condition, a condition it would have been impossible properly to estimate without the use of the sphygmomanometer. So he was advised that while a change in glasses might afford some relief, complete relief could not be looked for from that source, that the main cause of his trouble was elsewhere than in the eyes and that he had better consult with his physician again.

Being a man of means and of a systematic mind, he ordered new glasses as stated, in order to eliminate the possibility of the eyes causing any of his trouble, and proceeded to seek elsewhere for the cause of his symptoms, after expressing his deep appreciation of "the fairness and honesty" with which his inquiry at the writer's office had been met.

In a few days he returned and stated that he had had his physician, a dentist and a throat specialist make further examinations, with the result that the latter

lanced what appeared to be a perfectly healthy tonsil and obtained a quantity of pus therefrom. Immediate relief was obtained from some of his symptoms and he hoped to eventually secure full relief, but, of course, such a consummation is hardly to be expected in view of the several factors mentioned. In all probability, the infected tonsil is but one of many seats of the toxin.

Case 165 is another type of those who possess a normal systolic pressure, but who, nevertheless, cannot be accepted as normal in other respects. The patient, oddly enough, was sent by the patient whose case has just been given.

Nearly all the particulars of this case paralleled those in Case 184, under Class 3, Group B, the patient likewise being a young married woman of twenty-five. Her refraction was almost the same, her complaints similar, and the second, third and fourth phases of auscultation quite as indistinguishable as in that case. Her systolic pressure alone was different, being 132 mm. instead of 102 mm.

Great relief was experienced upon the wearing of

O. D. + 1. + .50 axis 170

O. S. + 1.75

but there still remained more or less occasional headache. Her esophoria of four degrees was left uncorrected for the reason that her hyperopia had never before been corrected and might, therefore, be the cause of its existence (perhaps temporary), and for the further reason that there were present evidences of toxemia. Her physician stated that a toxic condition of marked degree existed, and time would be required for its elimination. It

has been the writer's experience that these small amounts of esophoria may arise from such conditions and disappear coincidentally with the disappearance of the toxemia.

There also existed in this case more or less uncertainty regarding the full hyperopic defect. While the lenses given tended to blur distant objects, the patient could easily wear half a diopter more plus without perceptibly blurring the thirteen foot letters at fifteen feet. This characteristic is more fully dwelt upon under Diseases of the Urinary System.

The patient was dismissed with instructions to continue the treatment her physician had advised and to return to the writer in four or five months.

A third type is Case 171. This patient was a woman in the terminal stages of diabetes, yet it will be observed that her blood-pressure was practically normal.

She was fully cognizant of her condition but wished to learn if it was possible to relieve, with new lenses, her blurred vision and ocular pains. She was wearing lenses obtained only a few months before upon the following formula:

$$\begin{aligned} \text{O. D.} & - .50 \\ \text{O. S.} & - .75 - .50 \text{ axis } 180 \end{aligned}$$

A refraction indicated the need of

$$\begin{aligned} \text{O. D.} & - .25 \text{ axis } 80 \text{ 15/13 partly} \\ \text{O. S.} & - .50 - 1.50 \text{ axis } 165 \text{ 15/15 partly}^1 \end{aligned}$$

With the warning that these new lenses did not necessarily promise relief, the latter formula was made up for her and worn for two weeks. Upon her return, she reported great relief from the symptoms complained of, stating, however, that distant objects seemed more or

1. Subsequent refractions showed no change.

less distorted. As this was a natural sequence of the change in the axis of the left lens, she was assured that the condition would soon disappear, a prognosis which ultimately proved correct.

Now it was utterly out of the question to assure this patient that pleasing results could be confidently expected. Her condition was so extreme that not only was relief doubtful, but the occurrence of new developments of a disastrous nature would occasion no surprise. (Only a few days later the vision of the left eye was found to have been reduced to less than one-third. That of the right eye could go at any moment. The fundus had placed upon it hemorrhages at several points, being particularly numerous at the macula of the left eye. These were present at the first examination.) The lids drooped, indicating a partial ptosis, and claudication existed to a somewhat marked degree. It is evident, therefore, that disturbances of the ocular muscles might soon take place.

Yet the blood-pressure was normal except for the extreme faintness of the second, third and fourth phases which were hardly distinguishable, a fact which not only tends to prove the value of the latter sounds in auscultation, but which accentuates the necessity of making the examination of the patient as painstakingly as possible and observing all features of the case.

It so happened in this case that even neglecting the affection of the leg- and lid-muscles, there were unmistakable intraocular symptoms of general disease, but there may be cases in which such signs are lacking, and reliance must be placed upon instruments other than the ophthalmoscope and upon factors in symptomatology hitherto

neglected; for example, the condition of blood-pressure or of the urine.

These three types constitute the main exceptions of the general rule that *patients suffering from the ordinary symptoms of eye-strain, who have ocular defects sufficient to cause such symptoms, and who have a systolic blood-pressure ranging from 110 mm. to 145-150 mm., may confidently expect a large, perhaps a full, measure of relief by the wearing of such lenses as properly correct the ocular defects.* There are also other exceptions, but their occurrence is so seldom that it is hardly worth while to devote time and space to them. The practitioner learns, in time, how to differentiate, more or less accurately, between symptoms caused by ocular strain and those arising from other causes.

GROUP D

As stated, this group includes those whose systolic pressure is between 150 mm. and 170 mm. A division is hereby made among those which range above 150 mm., the reason for which will be seen shortly.

There has been some hesitancy about dividing this group into three classes, for the reason that the first class constitutes a very small number. Altho it is frequently true that lenses given a patient in this class accomplish the purpose desired, it is seldom that a blood-pressure of 150 mm. or over is observed without there being present some change in the fundus of the eye. This change from the normal may be slight, so slight that it would be overlooked if the observer was not very painstaking and constantly on his guard, or, on the other hand, the abnormality may be most pronounced, perhaps hemorrhagic or exudative in form. However, it may be

said that the latter forms are unusual, the hemorrhagic form especially, as it has been observed in only about two per cent of all cases examined. The exudative form is more frequent; altho no exact data have been gathered in regard to its frequency, it can safely be said to occur in about five per cent. of cases, perhaps more.¹

In view of these facts, it would not be profitable to devote great attention to

CLASS 1. When cases do occur within this class, they possess the same characteristics, practically speaking, as those in Class 1 in any of the other groups—the presence of refractive and muscular anomalies, the absence of the signs of other abnormalities, and relief from the symptoms complained of by the wearing of lenses.

CLASS 2, as in the other groups, consists of those cases in which there are symptoms of eye-strain, which lenses relieve, but in which there are also symptoms of other anomalous conditions—particularly ocular symptoms.

It is just about as easy to find a departure from normal on the fundus of an eye when the patient has a systolic pressure of 150 mm. or more as it is to find a refractive defect in the average individual. All practitioners are aware of such frequency. It is close to 100 per cent.

The condition which usually exists consists chiefly of alterations in the form and caliber of the vessels, and accentuation of the light streak along the top of the vessel walls, particularly the arterial wall. In regard to the alteration in form of the vessels, the arteries will

1. Reference is made particularly to the pronounced forms. The lesser forms occur much more frequently.

usually appear much more tortuous than is customary, a condition to be expected when there is considered the influence of increased pressure of a fluid upon the elastic tube containing it. They take a much more erratic course than is presented by the normal fundus, even more pronounced than that seen on the relatively flat fundus of the hyperopic eye.

The veins do not participate in this tortuosity so markedly. Both veins and arteries, however, may vary in caliber. Under such conditions, it is usual for either type of vessel to emerge from the optic disk in a somewhat constricted, narrow form;¹ to commence, at the edge of the disk, to swell in size and perhaps approach or equal *twice* its former size for two or four millimeters and then to gradually subside again.

Occasionally this variation takes place at the bend of a vessel, the latter increasing in size as the bend is approached and decreasing as it is passed. It would seem reasonable to suppose that this is caused by the impact of the blood against the wall of the vessel as it bends at a right angle, or more, to its former course.

The prominence of the light streak, particularly on the arterial wall, is a factor that must be noted, but more especially should it be observed if there is an apparent light streak of greater prominence than usual along the *side* of the vessels, particularly the veins. This streak differs from that usually observed, in that it does not appear and disappear according to the movements of the observer, as do so many of the light reflections from the fundus. It is a more or less constant condition and the

1. It is quite possible, often probable, that the vessel is only apparently narrowed at the disk, its full caliber being concealed by opaque tissue.

writer has noted its presence very often in cases such as are under discussion.

Mention of the effect of arterial pressure upon the veins should not be neglected. In this respect, there occurs one of the pathognomonic symptoms of beginning arteriosclerosis and it is vitally important to the patient that it be recognized.

Reference is made to the influence of the stiffened artery in depressing that part of the vein which may underlie it, in concealing it and in bending it in the direction of the arterial flow. In the first condition it will be found that the light streak of the vein is not apparently continuous—that it suddenly disappears a very short distance from the point at which the vessel passes under the artery. It reappears in the same manner on the other side of the artery, showing thereby that the vessel has been so depressed by the artery that its cylindrical surface no longer reflects light in the direction of the pupil.

In regard to the concealment of the vein by the artery, this is due, of course, to the opacity of the arterial wall caused by the advancing sclerosis.

The bending of the vein by the artery is caused by the tendency of the artery to elongate itself in response to the increased pressure, in consequence of which it pushes the vein in the same direction at the point where they come into contact, by reason either of its connection with the vein by means of strands of fibrous tissue or by the "friction-tight" condition imposed by the intraocular pressure. It is hardly possible that the arterial flow, *per se*, causes the bending of the vein, since the blood does

not come into contact with the vein, but it is, of course, true that indirectly the arterial blood column is the cause.

These comparatively minor changes constitute the premonitory signs of arteriosclerosis, and their observance by the refractionist gives him an unparalleled opportunity to fulfill the highest function conceivable in a work of this kind—the conservation of the general physical and ocular welfare of the patient as elaborated upon under General Considerations.

Careful study and appreciation of Arterial Pathology, Diseases of the Urinary System and Arteriosclerosis, as elucidated in this volume, reveal the immense importance of detecting these early signs of pathologic conditions, and of so advising the patient that he may obtain the proper medical attention, and be so instructed that the danger of an incipient disease becoming chronic is obviated.

It is not entirely a question of relieving or curing a patient for the time being; it is the great desirability of so directing the patient's actions and mode of living, etc., that he will not be afflicted in five, ten or twenty years with an incurable disease which may cause almost, or quite, intolerable misery—which might eventually lead to a partial or total blindness, or to a stroke of apoplexy, which either cuts short the patient's span of years or disables him for the rest of his life.

The onset of such diseases is so insidious and slow that the patient may be quite unaware of their presence until curative measures are undertaken too late. Only too often the patient expresses his belief that "he never felt

better," just before a cerebral hemorrhage renders further medical services unnecessary for all time.

A case of this kind occurred only recently among the writer's patients. A man of excellent physique came to him three years ago, before blood-pressure was being taken. Beyond an average refractive defect, nothing unusual was detected.

Two weeks ago the patient called for an adjustment of his glasses, stating at the time that they were still all right, and he did not feel the necessity of another examination. He was apparently in good health, appearing as well as could be expected for a man in the late fifties. Last week he suffered a stroke of apoplexy and died within a few hours.

Had blood-pressure observations been made at the examination three years ago, it is very probable that an abnormal reading would have been obtained, as the man undoubtedly suffered from the cause and result of hypertension, a condition which likely had been increasing for several years. He would have been properly advised in regard to the necessity of medical attention and probably would be a useful citizen today.

Cases similar to this occur with amazing frequency, as can readily be appreciated by study of the tables given in Chapter I, relative to the several principal causes of death throughout the country. Often it is brought home sharply by the sudden demise of a prominent member of the community, in the prime of life, or even of a member of our immediate families.

CLASS 3, consisting of cases in which lenses correcting ocular defects fail to relieve symptoms, is merely an accentuation of *Class 2*, the failure of lenses to give relief

being evidence of foreign diseases and supplementary to the hypertension and fundus-anomaly evidence. Whether the persistence of the local or remote symptoms is indicative of inherent weakness of the organ of vision, of the peculiar susceptibility of the ocular tissues to the toxic products of the disease or of the virulence of the latter, it is perhaps impossible to state categorically. Probably all three factors play their individual part separately or collectively, as the case may be.

Case 156 is one in point. This patient, a man of fifty-three, has been coming to the writer for many years. The formula of the lenses found necessary by an examination about a year and a half ago is:

O. D. - .25 - 2.50 axis $82\frac{1}{2}$ V. A. 15-13
 O. S. - .75 - 1.12 axis 100 V. A. 15-13

Decenter in 6 mm. each eye. (Three degrees of esophoria being present.)

Add + 1.50 O. U. for reading.

In August of this year (1915) he called and complained that he suffered frequently from local ocular pains after reading, and that after dinner in the evening he invariably was overpowered by a desire to go to sleep. Had the latter complaint alone been made, the writer might have been inclined to regard the case as one outside the optometric province, but its association with headache after reading threw a different light upon it.

A careful examination was made and the following formula written:

O. D. + .25 - 3. axis $87\frac{1}{2}$ V. A. 15-13
 O. S. - .75 - 1.50 axis 90 V. A. 15-13

Decenter in 6 mm. each eye. (Four and one-half degrees esophoria.)

Add + 2. for reading.

It was found, however, that at the reading point the esophoria disappeared and two degrees of left hyperphoria put in an appearance. There was also evidence of cyclophoria. The following lenses were made up for reading:

$$\begin{array}{l} \text{O. D.} + 2.25 - 3. \quad \text{axis } 87\frac{1}{2} \\ \text{O. S.} + 1.25 - 1.50 \quad \text{axis } 90 \end{array}$$

Decenter right lens 3 mm. up. Decenter left lens 4 mm. down.

There are several factors to be noted in this formula. First, there is the change in the spherical refraction of the right eye from $- .25$ to $+ .25$ without a similar change in the left eye. While such changes may occur occasionally, their occurrence is not sufficiently frequent to be regarded without interest and concern. Second, it will be noted that the astigmatism has increased at least half a diopter in each eye. The increase is really greater by nearly a quarter of a diopter, but the full correction was not given. Here, also, is a fact which frequently presents itself, but which, under the circumstances, may be regarded with suspicion. Third, the axes of the astigmatic defect have altered, the left to a marked degree. Fourth, the esophoria has increased. Fifth, hyperphoria has developed at the near point. Sixth, cyclophoria has appeared.

Taken separately in an ordinary case, not one of these changes merits great concern, but when considered collectively, there is justification for more or less alarm. This is particularly true of the muscle changes, as it clearly indicates degenerative changes along the course of the third, fourth or sixth nerve, perhaps in the cavernous sinus, which may be regarded as examples of changes that are taking place in other areas and which may result

in far more serious consequences, apoplexy, for example. Changes of this kind do not take place without reason, and the cause is usually deeply situated.

These fears were confirmed by the fundus picture in which there appeared a whitish area surrounding the macula (which later resolved itself into a mottled appearance); whitish lines, very faint, bordering the veins; a deposit of exudate at the junction of a vein with an artery, and, naturally, concealed veins where arteries passed over them.

Further confirmation was obtainable in the appearance of the skin below the lower lids, where it hung in very pronounced crescentic bags, but the final confirmation was found in the blood-pressure which registered: systolic 160 mm., diastolic 95 mm., pulse 65 mm.

These several factors pointed unmistakably to pathologic conditions arising primarily in areas other than ocular, and the patient was instructed to visit his physician at once. This was done, but the physician was given only one opportunity to make an examination, and that one revealed nothing of consequence other than a blood-pressure greatly in excess of the one discovered by the writer.

In view of the mass of evidence presented, there can be no question concerning the presence of pathologic conditions. The presence of albumin is in itself sufficient evidence of the accuracy of such a conclusion.

Whether simply renal, vascular, cirrhotic or cardiac or a complication of two or more of such diseases, the writer does not know, as such knowledge is beyond his province, but he is confident of the presence of something abnormal which was overlooked by the physician in

attendance. However, this oversight may have been quite pardonable in view of the single examination. Sometimes several examinations are necessary in order to establish the presence of, say, kidney disease.

As stated, the last mentioned formula was made up into lenses for reading, and the patient reported partial relief. Complete relief could hardly be expected, and the patient was so informed, it being stated at the same time that further visits to the physician were necessary. As these have not been made, the patient pleading pressing business (a fair example of the manner in which so many people neglect their duty to themselves), it cannot be expected that complete relief will take place. Indeed, more serious consequences may reasonably be regarded as imminent.

The ocular pain which resulted after close work had been done is no more than could be expected in view of the disturbance of the muscle equilibrium. Conditions which bring about such disturbances are certainly sufficient so to interfere with the metabolism of the muscle tissue that muscular effort results in pain.

Another example of unsuspected organic disease is found in

Case 79. The patient, a woman of fifty-nine, complained of temporal headache. If she had other symptoms, she did not see fit to mention them, or, at least, the writer can find no notes to that effect.

She was wearing

O. D. + 3.25 + .25 axis 165

O. S. + 3.50

Add + 2.50 for reading.

These lenses were obtained from the writer about four years previously.

The following formula was found necessary:

O. D. + 3.50 + .50 axis 165

O. S. + 3.75 + .25 axis 175

Add + 2.50 for reading.

It will be noted that the change is comparatively slight, not more than could be expected after an elapse of four years. However, the fundus picture was revealed as follows:

In the right eye the inferior temporal artery had a slight aneurysm which seemed to have a slight white spot in the center of it (perhaps a light-reflection only). The superior temporal artery twisted and doubled upon itself to a very marked degree.

In the left eye the inferior temporal artery was irregular in diameter, very tortuous and minus the light streak for a distance of 2 or 3 mm. from the disk. Otherwise, the light streak was rather pronounced upon all the arteries in each eye.

At the first examination, the blood-pressure was somewhat uncertain, as it varied from 150 mm. to 174 mm. At a subsequent one, after medical treatment, it subsided to 145 mm., as indicated in the table. Pulse rate was 90-92.

The patient was sent to a physician who reported chronic nephritis, and who instituted the treatment which resulted in lowering the blood-pressure as indicated.

Similar cases could be dwelt upon indefinitely, but these suffice to emphasize the necessity of those engaged in any form of eye work being able properly to recognize vascular changes which take place. A medical writer stated the situation only too truly when he asserted that

“not infrequently it is the man behind the ophthalmoscope who first discovers the evidence of the existence of these latter diseases” (nephritis, diabetes).

GROUP E,

in which the blood-pressure ranges above 170 mm., presents the most marked of ophthalmoscopic evidence. It is among such cases that the beautiful examples of choked disk, retinitis albuminurica, retinitis hemorrhagica, silver-wire arteries, etc., are found, thus presenting evidence which is unmistakable even without the aid of sphygmomanometric findings.

In this group, it is obvious that

CLASS 1 is practically non-existent. It is almost an impossibility for hypertension of this degree to exist without producing fundus changes so marked that even a casual examination reveals them. This is particularly true if the hypertension is sustained for a considerable length of time. In acute cases where the hypertension is only temporary such changes may have had no time to develop.

CLASS 2, which includes cases that obtain relief from symptoms complained of by the wearing of glasses, but which present objective evidence—ophthalmoscopic or otherwise—of pathologic conditions, is likewise a limited class. It is true, however, that such individuals sometimes request glasses only for the improvement of vision, distant or nearby, and do not complain of other symptoms.

When there have occurred no lesions so great or so situated as to cause the defective vision, and lenses fully

supply the patient's expressed wants, the case naturally falls into this class. Such, for example, is

Case 73. This patient, age fifty-four, complained that his glasses were not satisfactory, particularly in reading.

He was wearing for all purposes

O. D. — 1. — 1. axis 90

O. S. — .50 — .75 axis 105

He was found to require

O. D. — 1.25 V. A. 15-15

O. S. — .25 — .50 axis 80 V. A. 15-15

Add + 1.75 for reading.

These lenses were made up and found very satisfactory.

It will be noted that his blood-pressure was 180 mm. The net-work of choroidal vessels was naturally very pronounced, owing to the myopic condition, and the edges of the optic disk were very irregular, a natural sequence of the myopia. No other marked irregularities were observed (according to the records).

There were certain reasons why it was considered inadvisable to suggest medical services to this patient, and the failure to do so was further justified by the complete satisfaction derived from the glasses.

Similar cases occur from time to time and must be handled in accordance with the individual circumstances. Usually it is but a question of time before the patient will suffer unpleasant symptoms of the condition which actually causes the elevated tension, and when the situation is such that the advice to seek medical attention is appropriate, it should be offered as a matter of course. There are times when such action on the part of the patient must be insisted upon. For it is true that occa-

sionally extremely high pressures are encountered without the patient knowingly suffering symptoms of the condition, and unless thoroughly impressed with the necessity of medical attention, the neglect to remedy the fault will continue.

CLASS 3 includes the cases in which there is failure to obtain partial or total relief by means of lenses. The symptoms may consist of vertigo, headache, eye-ache, digestive disturbances or blurred vision. The latter, especially in monocular form, is particularly frequent and is due, usually, to the destruction of the optic nerve fibers, either in their course in the nerve sheath or in their retinal endings. The latter seems to be the more frequent. The best example of such a condition is a case previously mentioned by the writer elsewhere.¹

Case —. A woman of fifty-three years, who had been given bifocal lenses a short time before the writer was taking blood-pressure regularly, called and complained that the vision of the left eye had suddenly been greatly reduced in acuity.

She was wearing the following formula, which, when prescribed, had given the vision indicated:

O. D. + .25 — 1.25 axis 155 V. A. 15-20 partly.

O. S. + .25 — .75 axis 20 V. A. 15-13 partly.

A test showed the vision of the left eye now to be 15-45; the right eye remained the same as at the previous examination.

The ophthalmoscope revealed several hemorrhages in both eyes; sclerosed arteries in each eye, as evidenced by the light streak on the arteries and the depressed veins

1. Optical Journal and Review, July 1, 1915.

as the arteries crossed them, and a great disturbance in the macular region of the left eye, there being masses of pigment in evidence, and albumin to a marked degree.

The systolic blood-pressure was found to be 210 mm., the diastolic 116 mm. Immediate medical attention was advised, and this advice was followed. The physician verified the high pressure, found evidence of an advanced chronic nephritis and sent the patient home to bed with the remark that she "was ready to blow up." Treatment was instituted and resulted in improvement, tho it cannot be said that prognosis is particularly favorable, as the case was too far advanced. The patient later called at the writer's office and thanked him for "certainly saving her life."

Case 240 may also be submitted. The patient, a woman of fifty-nine, came to the author for the first time but a month previous to this writing. She complained of blurred vision, both for distance and reading; pain over the eyes, and attacks of vertigo.

A refraction gave the following results:

O. D. + 1. V. A. 15-13 partly.
O. S. + .75 V. A. 15-60
Add + 2.50.

The ophthalmoscope revealed macular deposits, depressed, concealed and tortuous veins (almost a complete loop being formed at one point), arteries pale and lined with white tissue, probably vascular opacity.

Blood-pressure was 240 mm. and the patient was advised to consult a physician at once. This was done, the physician listened to the patient's complaint, heard her state that the writer had found an abnormal blood-pressure, made practically no examination at all and

dismissed her with the information that there was nothing wrong (*sic*) but a little stomach trouble, for which he gave her a simple remedy!

The patient reported her experience to the writer, who then instructed her to repeat her call upon the physician. In the meantime the latter was called up and informed of the ocular condition and of the state of blood-pressure, concerning which he had not taken the time and slight trouble to ascertain, and it was requested that he make a more thorough examination.

This was done at the patient's next call *and the presence of kidney disease established!*

It is just such experiences as this which prompt the skepticism expressed in Case 156 of the preceding group. There is absolutely no excuse for a physician dismissing as inconsequential such a case as the one cited. There may be occasionally a reason for not acquainting the patient with the full gravity of his or her condition, but at least a warning can be uttered; if not to the patient, then to a member of the family—and it may be added that the woman's daughter accompanied her to the physician's office.

Case 53 is not essentially different. The patient, a man of fifty-eight, hair white, physique average, general color rather pasty white, wanted different glasses for his desk work, as his present ones blurred for that distance, being too strong. He stated that he knew he was in poor health.

A refraction revealed:

O. D. + 1.25 + .50 axis 165 V. A. 15-20

O. S. + 1.25 + .50 axis 50 V. A. 15-15 partly.

Add + 2. for reading.

The refraction of this case presented the usual characteristics of high blood-pressure—uncertain response to

reduction or increase of plus lenses, and decreased visual acuity without there being present a perceptible haziness or lesion of the retina. The only visible condition of the fundus that could be considered abnormal was the appearance of the optic nerve. The outer border of the disk was a natural pinkish white. The middle area surrounded by this border was an extremely dark gray.

In less marked forms, this condition exists with sufficient frequency to be classed as a physiological variation. In this case, however, the differences in color were so accentuated as to arouse suspicion.

Otherwise, as stated, the fundus was normal. The arteries had no visible markings of disease and, as they crossed the veins, there was no depression of the latter as is usually found when the blood-pressure is high or the arteries sclerosed.

Altogether, if action other than optometric in this case rested entirely upon the ophthalmoscopic picture, the usual procedure would be to suspend judgment, observe the case for a certain length of time and finally advise according to developments. The result would be not altogether favorable to the patient.

Having finished with the ophthalmoscope, the sphygmomanometer was applied. A systolic pressure of 175 mm. and a diastolic of 90 mm. were registered. The patient was asked if he was occasionally affected with dizzy spells, and the reply was in the affirmative. A further admission was obtained to the effect that a urinalysis some three years before had disclosed a bad condition but that nothing further was done about it.

Even without the ophthalmoscopic and sphygmomanometric evidence, it was plain to be seen, from both the general appearance and subjective symptoms, that

the patient was a sick man, yet when he asked a physician to examine him the latter looked at him, felt his pulse and *without further examination pronounced him a well man!* Is it any wonder that the layman sometimes is skeptical of medical diagnosis?

It can be stated, with more or less confidence, that patients presenting evidence such as is found in this class of Group E are in the last stages of the disease which causes such radical conditions. It should always be remembered that the vascular system of the eye is a terminal system and one representative of similar systems situated in the kidneys and, particularly, in the brain. When extensive changes take place in the retinal vessels, causing a thickening of the walls of the arteries and permitting the escape of albumin in sufficient quantities to be seen ophthalmoscopically—even permitting the rupture of the vessels, as takes place in retinitis hemorrhagica—it can safely be assumed that similar changes are taking place in the cerebral or renal vessels, or perhaps in the cardiac vessels, which will ultimately result in apoplexy or heart failure.

The case has become a serious one, and the most expert medical attention is necessary in order that the patient's life may be saved or serious illness averted. Even then the prognosis is not very favorable in the majority of cases. All too often, death occurs before five or ten years have elapsed.

The student has now been led through all the stages of blood-pressure that may be encountered in optometric practice. He has been advised concerning the ocular conditions that can be expected in each instance. It has been pointed out that low pressures are more particularly

associated with cases in which local irritability persists—in which there is more the evidence of exhaustion than of the breaking down of structural tissue; that normal pressures do not necessarily mean freedom from pathologic conditions, tho the percentage of cases possessing pathologic conditions coincident with such a pressure is less than otherwise; that the moderately high pressures introduce a more or less different form of local irritability than that brought about by a low pressure, in that it is a more positive pain, rather than the weariness and discomfort due to exhaustion; that the high pressures are associated with the destruction of tissue and the profuse liberation of the toxic products of metabolism, resulting in conditions not to be relieved by the wearing of glasses, such as amblyopia—frequently very marked—local pains, headache, nervousness, indigestion, etc.

It is the writer's hope that all points are made with sufficient clarity to be understood. It is a matter of course that a subject so great as this one cannot be adequately encompassed by the covers of a book, and so it is to be expected that proficiency in the application of sphygmomanometry in optometric work can be attained only by practice, a fact which encourages the belief that points which may now be obscure to the reader will be made clear when sufficient experience has been obtained.

The inclusion of this comparatively new science in the writer's system of ocular examination has led to enormously satisfactory results. It will give the reader some idea of the protection it has offered patients, both in regard to general health and ocular health, when it is stated that, *previous* to its employment, not more than five per cent of cases were sent to the general medical practitioner for treatment, while *since* its employment, *not*

less than twenty per cent have been so directed. Their ills have ranged from those of minor character to those which lead to an early and complete collapse. The writer is confident that, by so doing, not only have many eyes been preserved for future normal vision, but that many years have been added to the lives of a large number of his patients.

In respect to the protection it offers the examiner, that seems too obvious to merit greater consideration than is offered in Chapter I. It is absurd to expect to relieve the symptoms of eye-strain in each case that presents itself, owing to their simulation by the symptoms of anomalies other than ocular. It therefore becomes necessary to be able to determine when such relief can, or cannot, be expected by the wearing of glasses. In this respect, sphygmomanometry is of inestimable value as it yields information obtainable in no other way of which the writer is aware.

There is, therefore, no doubt in the writer's mind concerning the future of the science in optometric work. It so adequately clarifies so many of those cases which have remained obscure mysteries, much to the discomfiture of the practitioner—and discomfort of the patient—that it will be welcomed by those who understand and appreciate it as meriting a greater evaluation than many of the instruments now utilized and considered indispensable.

NOTE I.

Mr. Hunter cites the effect of alcoholic beverages when indulged in habitually, tho not necessarily often to excess. He is quoted as follows:

“Nothing has been more conclusively proved than that a steady free use of alcoholic beverages, or occasional excesses, are detrimental to the individual. In my judgment, it has also been proved beyond peradventure of doubt that total abstinence from alcohol is of value to humanity; it is certain that abstainers live longer than persons who use alcoholic beverages.

“Among the men who admitted that they had taken alcohol occasionally to excess in the past, but whose habits were considered satisfactory when they were insured, there were 289 deaths, while there would have been only 190 deaths had this group been made up of insured lives in general. The extra mortality was, therefore, over fifty per cent, which was equivalent to a reduction of over four years in the average life of these men. If this meant that four years would be cut off the end of the average normal lifetime of each man, there are many who would consider that ‘the game was worth the candle’; but it means that each year a number of men will die at an earlier date than they should. For example, at age thirty-five, the expectation of life is thirty-two years; in the first year after that age, instead of, say, nine persons dying, there would be probably twelve deaths; that is, three men would each lose thirty-one years of life, etc. As a matter of fact, many immoderate drinkers

would live longer than thirty-two years, but not nearly so many as would live if they had been moderate drinkers, and far fewer than if they had been total abstainers from alcohol.

“With regard to men who had used alcoholic beverages daily, but not to excess, the experience of the companies was divided into groups: (a) men who took two glasses of beer, or a glass of whiskey, or their equivalent, a day; (b) men who took more than the foregoing amount, but who were not considered by the companies to drink to excess. The mortality in the second group was found to be fully fifty per cent greater than in the first—an excellent argument for moderation in the use of alcoholic beverages. The foregoing result does not mean that the large excess mortality in Class (b) was due to their drinking a little more each day than those in Class (a). It is probable that among those who were very moderate users of alcoholic beverages there were comparatively few who eventually used liquor immoderately; but among those who took more than a glass of whiskey or its equivalent a day, there were probably a goodly number who increased their daily consumption after having applied for insurance, and who eventually drank to an immoderate extent. Part of the hazard from alcoholic beverages lies in the user losing the power to limit himself to a moderate consumption.

“Among the men whose habits were formerly intemperate, but who had reformed for at least two years prior to their acceptance by the insurance companies, the extra mortality was fully thirty per cent; *i.e.*, their average lifetime was reduced by about three years. This excess mortality is partly due to the effect of previous intemperate habits in undermining the system and partly

to a proportion of the persons relapsing into their old habits."

Another example cited relates to syphilis. His own words are again given:

"It has generally been assumed that syphilis increases the death rate, but no facts relating to this country have heretofore been published showing the serious effect of that disease on mortality. The forty-three companies accepted only the better type of cases with a history of syphilis. Taking all the cases in which the insured admitted having syphilis prior to the date of application for insurance, the companies had 274 deaths, while the normal mortality would have been about 153; that is to say, there was an extra mortality of about eighty per cent due to that disease. The same results have been found in other countries. For example, among the men insured with the principal German company, there were 487 deaths among those who had undergone treatment for syphilis, while there would have been only 290 deaths according to the experience of that company among their insured lives; the excess mortality was nearly seventy per cent. The only other published investigation of which I am aware was that made by the Scandinavian companies, which showed a mortality as high as in the German company to which I have just referred. This is sufficient evidence of the bad effect of syphilis on the individual. It is hardly necessary to mention that, in addition to shortening men's lives, it shortens their period of activity while they are alive. Locomotor ataxia and softening of the brain are believed by medical men to be due to syphilis in the majority of cases.

"It must not be assumed that the high mortality is due to the companies accepting cases which had not been

properly treated for syphilis. One of the classes consisted of men who had had two years' continuous treatment and one year's freedom from symptoms prior to acceptance by the insurance companies, yet the excess mortality was eighty per cent—a decreased lifetime of about six years. A disease which shortens the life and limits the activity of the individual, and which causes untold misery and suffering to the progeny, should be and can be banished by an intelligent public opinion."

Mr. Hunter states that about two hundred classes or groups were investigated, and he gives other examples, but the two above presented suffice to establish the contention.

The principles involved apply, of course, to all preventable diseases. Life can be prolonged if the causes of such disease are eradicated and the individual be influenced so to conduct himself that he is neither the source nor the recipient of such organisms that lead to the establishment of pathologic areas in his physical economy.

Nevertheless, while this truism is self-evident, its practice is too limited to show, at the present time, conclusive results susceptible to demonstration by other than the microscopic scrutiny of scientific analysis. The fact that life has been lengthened by an average of fourteen years in the last hundred cannot be accepted as such evidence, for the simple reason that the gain has been made largely in the prevention of infant mortality.¹ For example, if one individual dies at sixty-four and another at two, the average life for the two is thirty-three years. If, on the other hand, both die at forty-seven, altho

It is a matter of pardonable pleasure to the author that since arriving at the above conclusion independent of medical writers, it has been noted that the same conclusion is arrived at by no less an authority than L. M. Warfield, (See Arterio-Sclerosis, L. M. Warfield, C. V. Mosby Co., St. Louis, Mo.)

there is an average gain of fourteen years, it is an open question whether the forty-five years added to the one compensates for the seventeen lost by the other, since the one period of time is spent largely in gaining experience, while the other is spent in applying it, or, in other words, in fructifying. The value of saving the child is curtailed if the man is permitted to come to an untimely end.

The fact that it is impossible to estimate, with any reasonable degree of accuracy, just how long a certain individual will live if he follows prescribed rules, while it is quite possible to make a similar estimation of a group of individuals, indicates that while the laws of cause and effect are more or less definitely known when applied generally, the result of their application in the individual is not so assured. For example, one may rigidly adhere to a manner of living determined to be the best by the most thorough scientific investigation, and yet die comparatively young, while another, who breaks all prescribed rules, will live to a ripe old age.

It is precisely this apparent contradiction of the law of consequences that encourages men to undertake, or continue, the violation of Nature's rules. They hope to escape the consequences. One can point out bad examples without number, and still the average individual will not take the lesson to himself. He will take a chance. Statistics mean nothing to him. He must learn by bitter experience that he is not immune from the laws of cause and effect.

The unfortunate feature is that after having had this bitter experience, there is no possibility of recovering lost ground. As Mr. Hunter's figures so well demonstrate, even the reformed drinker—the same reasoning can be

applied to the reformed eater or to anyone who has indulged in excess of any kind tending to poison the system or deplete the resisting power—dies earlier than the average, clearly indicative that health is analogous to the bank account; it cannot be spent and retained too.

Yet the average person gives only passing thought to this philosophy. Indulgence in one form or another is so common as to be taken as a matter of course. The inevitable result is a premature demise, a reflection of which is found in vital statistics where there is a preponderance of deaths due to the diseases that are clearly evidences of excesses in one form or another, or of negligence.

NOTE 2.

For a period, the writer was inclined to believe that the sound in the first phase was caused by the striking together of the flattened walls of the arteries *after* the wave had passed through and the vessel had returned to its former compressed position. Investigation, however, disclosed the fact that the sound as heard with the stethoscope, occurred coincidently with, or perhaps slightly in advance of, the beginning of the oscillation of the hand on the dial of the sphygmomanometer, indicating that the tone vibrations of the arterial walls took place at the height of the systolic wave. If, on the other hand, the dial-hand oscillated *before* the sound-phenomena had resulted, it would have rather definitely proven that the walls first expanded and then contracted before the sound resulted, which would justify the surmise that the *contraction* of the arterial walls together was the cause of the sound, and not their expansion.

It might be thought that the time consumed in transmitting the sound waves would be different from that

required for the transmission of the air-compression waves to the sphygmomanometer, but multiplying the difference between the distance of the stethoscope ear-pieces from the armlet and that of the sphygmomanometer seemed in no wise to alter the circumstances, as it necessarily should if this thought was based upon known laws of physics. Second thought, however, makes it clear that air-compression waves and sound waves are one and the same, and travel with precisely the same speed. Altering the distance as above described will make no perceptible difference between the registration of the air-compression waves by the sphygmomanometer and the registration of the sound waves on the ear drum. As sound waves travel at the approximate rate of 1,100 feet per second, the difference in the lengths of the tubing would have to be several hundred feet before a difference in registration would be physiologically detectable.

GLOSSARY¹

A

ABDUCENS, or sixth nerve. Nerve supplying the external rectus muscle.

ABCESS. A circumscribed cavity containing pus.

ACCOMMODATION. Adaptation; adjustment.

A. of the EYE, the power to adjust the crystalline lens that its focusing value may meet the requirements imposed by objects situated at variable distances, to the end that the image of the object viewed may be accurately focused upon the retina of the eye.

ADRENALIN. The active principle of the suprarenal gland.

ALBUMIN. A proteid, animal or vegetable, which is soluble in water and coagulable by heat.

ALBUMINURIA. An excess of albuminates in the urine.

AMAUROSIS. Partial or total blindness.

AMBLYOPIA. Dimness of vision.

AMETROPIA. Abnormal refraction of the eye.

AMPLITUDE. The range or extent, as of the pulse or of the accommodation of the eye.

AMPULLIFORM. Flask shaped; dilated.

ANABOLISM. Constructive metabolism.

ANASTOMOSIS. The junction of vessels or hollow organs.

ANATOMY. The science of organic structure.

ANEMIA. A deficiency of blood or red corpuscles.

A., **PERNICIOUS**, that caused by disease of the blood or of the blood-making organs.

ANEROID. Dispensing with fluid.

ANESTHESIA. A state of insensibility.

ANEURYSM. A dilatation of an artery.

ANOMALY. That which deviates from the ordinary.

ANOREXIA. An absence or a loss of the appetite.

AORTA. The main arterial trunk.

AORTIC REGURGITATION. A flowing backward of the blood through the defective valves of the left ventricle.

1. Practically all of these definitions have been obtained from Gould's Pocket Medical Dictionary. The reader is urged to purchase that little volume, as it contains a very comprehensive list of medical words.

APOPLEXY. Paralysis from rupture of a cerebral, or other, vessel.

APPENDAGE. That which is attached to an organ as a part of it.

APPENDICITIS. Inflammation of the appendix vermiformis.

ARTERIOLE. A small artery.

ARTERIOSCLEROSIS. The hardening of the arterial walls.

ARTERY. A vessel carrying blood from the heart.

CENTRAL RETINAL, artery supplying the retina of the eye.

ASTHENOPIA. Weak or painful vision.

ATMOSPHERE. A unit of pressure, fifteen pounds to the square inch.

ATROPHY. A wasting of a part from a lack of nutrition.

AURAL. Pertaining to the ear.

AUSCULTATION. A method of determining the condition of an organ by listening to the sounds produced by it.

AUSCULTATORY. Pertaining to auscultation.

AUTOINTOXICATION. A morbid condition produced by poisonous products elaborated within the body.

AXIAL, AXILE. Pertaining to an axis.

B

BLADDER. The sac-like receptacle for the urine.

BRACHIAL. Pertaining to the arm.

B. ARTERY, principal artery of the upper arm, running beside the humerus bone.

BRADYCARDIA. (Same as Brachycardia.) Abnormal infrequency of the pulse.

BRIGHT'S DISEASE. A generic term for acute and chronic diffuse disease of the kidneys, usually associated with dropsy and albuminuria.

C

CALISTHENICS. A system of light gymnastics.

CANCER. A malignant tumor with the production of epithelioid cells.

CAPILLARY. A minute blood-vessel.

CARDIAC. Pertaining to the heart or cardia.

CAROTID. The principal artery of the neck.

C. PLEXUS, the nerve-plexus around the carotid artery.

CARTILAGE. Gristle; a nonvascular elastic tissue softer than bone.

CATARACT. Opacity of the crystalline lens.

- CATARRH. Inflammation of a mucous membrane.
- CAVERNUS. Having hollow places.
C. SINUS, a sinus on the body of the sphenoid.
- CEREBRAL. Relating to the brain.
- CERVICAL. Pertaining to the neck or to a cervix.
- CHLOROSIS. A form of anemia most common in young women, marked by greenish color of the skin and menstrual disturbances.
- CHOKED DISK. Inflammation of the papilla of the eye. Optic neuritis.
- CHOLESTERIN. A monatomic alcohol, found in blood, nerve-tissue, and bile.
C. CRYSTALS, sometimes found in the vitreous humor of the eye. When viewed ophthalmoscopically, they resemble a shower of minute, shining flakes of gold.
- CHOROID. The second or vascular tunic of the eye.
- CHOROIDITIS. Inflammation of the choroid.
- CILIARY. Pertaining to the cilia (hair-like processes of certain cells).
C. GANGLION, the ganglion at the apex of the orbit.
C. MUSCLE, the muscle of accommodation of the eye.
- CILIORETINAL. A small retinal artery leading from the optic disk to the macula.
- CIRCULATION. The passage of blood through the body.
C., COLLATERAL, that taking place through secondary channels after stoppage of the principal route.
- CIRRHOISIS. Hardening due to an increase in the connective tissue of an organ.
- CLAUDICATION. Lameness.
- COCAIN. An alkaloid from coca; it is a powerful local anesthetic, and internally is used as a narcotic.
- CONJUNCTIVA. The mucous membrane of the eye.
- CONSTIPATION. A sluggish action of the bowels.
- CONSTRICTOR. A contracting or compressing muscle.
- CORNEA. The transparent anterior part of the eyeball.
- CORTICAL. Pertaining to the cortex, which is the external gray layer of the brain.
- CRANIAL. Pertaining to the cranium.
- CROOKES GLASS. A neutral colored glass designed especially to absorb a maximum amount of both heat and ultra-violet rays.

CRYSTALLINE. Like a crystal.

C. LENS, the transparent lens of the eye.

CYANOSIS. Blue discoloration of skin from nonoxidation of blood.

CYCLITIS. Inflammation of the ciliary body.

CYCLOPHORIA. A tendency of the eye to twist around its antero posterior axis.

CYCLOPLEGIA. Paralysis of the ciliary muscles.

D

DACRYOCYSTITIS. Inflammation of lacrimal sac.

DEGENERATION. Deterioration in structure of a tissue or organ.

D., AMYLOID, characterized by the formation of an albuminous substance, resembling starch in chemic reaction.

DEGLUTITION. The act or power of swallowing.

DELIRIUM. Mental aberration due to disease.

DIABETES. A disease characterized by an excessive flow of urine.

DIAGNOSIS. The recognition of a disease from its symptoms.

According to Webster, "The art or act of recognizing the presence of disease from its signs or symptoms, and deciding as to its character; also, the decision arrived at.¹ Critical perception or scrutiny; judgment based on such scrutiny. Scientific determination of any kind."

D., DIFFERENTIAL, the distinguishing between diseases with similar symptoms.

DIARRHEA. Morbidly frequent evacuation of the bowels.

DIASTOLE. The period of dilatation of the heart.

DILATATION. An expansion of a vessel or an organ.

D. of HEART, an increase in size of one or more of the heart cavities from weakening of the muscles.

DILATOR, DILATATOR. An instrument for stretching a cavity or opening; also a dilating muscle.

1. It seems to the writer that this definition covers the act of concluding that, in an individual case, the headache or other indisposition from which the patient suffers is caused by eye-strain. It also applies to the decision, in an individual case, that blurred vision is due either to refractive anomalies or to pathological conditions, as the case may be. It is the writer's opinion that optometrists *do* diagnose, altho it is not necessarily true that they attempt, or are justified in attempting, to render a differential diagnosis in a case of ocular or other diseases, *i.e.*, determine the difference between, for instance, retinitis and choroiditis. The active, inquisitive mind can hardly help diagnosing a condition (judging, forming an opinion about) but, in the circumstances under consideration, conclusions concerning the *kind* of disease that may be present should not be advanced by the optometrist. In other words, a *mental* diagnosis may be unavoidable, but it should not be asserted. Even under pressure of the patient's inquiries, only a most guarded opinion is permissible, perhaps never a positive statement.

DISC, DISK. The papilla.

DISTAL. Peripheral; away from the center.

DUBOISIN. An alkaloid from *Duboisia myrporoides*.

DURA MATER. The outer membrane of the brain and spinal cord.

DYSPEPSIA. Impaired or imperfect digestion.

E

ECZEMA. Inflammation of the skin with exudation of lymph.

EDEMA. Accumulation of serum in the cellular tissue.

ELIMINATION. Excretion.

EMBOLISM. The obstruction of a blood-vessel by an embolus.

EMBOLUS. A blood-clot or other body carried by the blood current and obstructing circulation at the point of lodgment.

ENCEPHALOPATHY. Any disease of the brain.

ENDARTERITIS. Inflammation of the intima of an artery.

ENDOCARDITIS. Inflammation of the endocardium.

ENDOTHELIUM. Lining membrane of vascular and serous cavities.

ENOPHTHALMOS. Retraction of the eyeball from spasm of the extrinsic eye-muscles.

EPILEPSY. A nervous disease with loss of consciousness, and tonic and clonic convulsions.

EPIPHORA. An overflow of tears.

EPISTAXIS. Hemorrhage from the nose.

ESOPHORIA. The tending of the visual lines inward.

ETIOLOGY. The science of the causes of disease.

EUPHOS GLASS. A greenish-yellow glass designed to eliminate the ultra-violet rays. It is very similar to Fieuzal glass.

EXCITATION. The act of stimulating or irritating.

EXOPHTHALMOS. Abnormal protrusion of eyeballs.

EXOPHORIA. The tending of the visual lines outward.

EXTRAVASATION. An effusion of fluid into the tissues.

EXTRINSIC. External, outward.

EXUDATE. The product of exudation.

EXUDATION. The morbid oozing out of fluids.

F

FACIAL. Pertaining to the face.

FENESTRATED. Having apertures or openings.

FIBROUS. Consisting of or pertaining to fibers.

FILAMENT. A thread-like structure.

FLEXURE. A bending.

FOVEA. A small fossa or depression.

F. centralis retina, a small depression in the macula lutea.

FUNCTIONATE. To perform a function; to act; operate.

G

GANGLION. 1. A semi-independent nervous center.

2. An enlarged lymphatic gland.

G., CERVICAL, SUPERIOR, that opposite the second and third cervical vertebrae.

GASEOUS. Of the nature of gas.

GASTRIC. Pertaining to the stomach.

GLAUCOMA. A disease of the eye, characterized by increased intra-ocular tension.

GLOSSOPHARYNGEAL. Pertaining to the tongue and pharynx.

GRANULAR. Composed of grains or granulation.

H

HEART. The hollow muscular body, the center of the circulatory system.

HEMERALOPIA. Night blindness.

HEMIANOPSIA, HEMIANOPIA. Blindness of one half of the visual field.

HEMORRHAGE. A flow of blood from the vessels.

H., SUBCONJUNCTIVAL, a hemorrhage beneath the conjunctiva.

HEPATIC. Pertaining to the liver.

HERNIA. The protrusion of a viscus from its normal position.

HG. Hydrargyrum (mercury).

HISTOLOGY. The study of the intimate structure of tissues.

HOMATROPIN. An artificial alkaloid from atropin; it is used as a cycloplegic.

HUMERUS. The large bone of the upper arm.

HYDRODYNAMICS. The principles of dynamics as applied to water and other liquids.

HYDROSTATICS. That branch of science which relates to the pressure and equilibrium of nonelastic fluids, as water, mercury, etc.

HYPEREMIA. Excessive amount of blood in any given part of the body.

HYPEROPIA. "Far-sightedness." Condition in which parallel rays of light focus behind the retina of the eye when the ciliary muscle of the eye is at rest.

HYPERPHORIA. A tendency of the visual axis of one eye to be above that of the other.

HYPERTENSION. Tension that is too high.

HYPERTROPHY. Abnormal increase in the size of a part or an organ.

HYPOPYON. Effusion of pus in the anterior chamber of the eye.

HYPOTENSION. Tension that is too low.

HYSTERIA. A functional necrosis with abnormal sensations, emotions or paroxysms.

I

IDIOPATHIC. Spontaneous; primary.

IMPACT. To drive close; to press; wedge, or drive so as to fix firmly.

INFECTION. The communication of disease germs.

INFILTRATION. A fluid effusion into an organ or a tissue.

INFLUX. An inflow.

INGESTION. The introduction of food into the body.

INSOMNIA. Inability to sleep.

INTIMA. The innermost coat of the vessels.

IRIDII. Plural of iris, which is the colored membrane of the anterior part of the eye.

IRITIS. Inflammation of the iris.

J

JAUNDICE. A yellow coloration of the skin.

K

KATABOLISM. The retrograde chemic changes in the tissues of the body.

KERATITIS. Inflammation of the cornea.

KIDNEY. The organ secreting urine.

L

LAGOPHTHALMUS. An inability to close the eyes.

LESION. Structural tissue-change from injury or disease.

LETHAL. Deadly; fatal; causing death.

LEUKEMIA. A fatal blood disease with a great increase in the number of white corpuscles.

LOCOMOTOR. Relating to locomotion.

L. ATAXIA, a disease of the posterior columns of the spinal cord, marked by fulgurant pains, disturbances of sensations, etc.

LUES. Syphilis.

L., CONGENITAL, existing from birth; innate.

LUMEN. The cavity of a tubular structure.

M

MACROSCOPIC. Visible to the naked eye.

MACULA. The yellow (most sensitive) spot of the retina.

MASTICATION. The process of chewing.

MAXIMAL. Greatest; in this connection it is used as a substitute for systolic.

MEDIA. The middle coat of a vein, artery or lymph vessel.

MENINGEAL. Pertaining to the membranes of the brain and cord.

MERCURY. Hydrargyrum; a white, heavy, liquid metal.

METABOLISM. A change in the intimate condition of cells, constructive or destructive.

METASTATIC. Pertaining to metastasis, which is a change in the seat of a disease.

MINIMAL. The least; in this connection it is used as a substitute for diastolic.

MORBID. Pertaining to disease.

MORBIFIC. Causing disease.

MUCOUS. Having the nature of mucus.

M. MEMBRANE, the membrane lining those cavities and canals communicating with the air.

MUSCAE VOLITANTES. Floating spots in the visual field.

MUSCULAR. Pertaining to muscle.

MYDRIASIS. Dilatation of the pupil.

MYOPIA. "Near-sightedness." Condition in which parallel rays of light focus in front of the retina when the ciliary muscle of the eye is at rest.

MYOSIS. Abnormal smallness of the pupils.

N

NECROSIS. The death of a circumscribed piece of tissue.

N., COAGULATIVE, a form marked by formation of fibrin.

NEGATIVE. The opposite of positive.

NEPHRITIS. Inflammation of the kidney.

N., INTERSTITIAL, that involving the connective tissue.

N., PARENCHYMATOUS, that involving true renal parenchyma.

NEURAL. Pertaining to nerves.

NEURALGIA. Pain in a nerve.

NEURASTHENIA. Exhaustion of nerve-force.

NEURITIS. Inflammation of a nerve.

N., RETROBULBAR, that of the optic nerve posterior to the eyeball.

NICTATION, NICTITATION. The act of winking.

NUCLEAR. Pertaining to the nucleus.

NUCLEUS. The essential part of a typical cell and the controlling center of its activity. The controlling center of a muscle or organ.

NYCTALOPIA. Day-blindness.

NYSTAGMUS. Oscillatory movement of the eyeballs.

O

OBJECTIVE. Pertaining to things lying external to one's self.

OCCIPITAL. Pertaining to the occiput, which is the back part of the head.

OCULOMOTOR. Pertaining to eye-movements, or to the third nerve.

OPHTHALMIA. Inflammation of the conjunctiva.

OPHTHALMIC. Pertaining to the eye.

O. ARTERY, the artery supplying the orbit and its contents.

OPHTHALMOPLÉGIA. Paralysis of the ocular muscles.

OPTIC. Pertaining to vision or its organ.

O. DISC, the entrance of the optic nerve into the retina.

O. FORAMEN, the sphenoid opening for the optic nerve.

ORBICULARIS. A name given to muscles whose fibers encircle an orifice.

ORBIT. The bony cavity of the eyeball.

ORIENTATION. The location of one's position in a given environment.

OSCILLATION. A swinging or vibration.

OSCILLATORY. Moving, or characterized by motion, backward and forward; swinging.

P

PACHYMENINGITIS. Inflammation of the dura.

PALPATE. 1. To explore with the hand.

2. Having tactile organs.

PALPITATION. Violent pulsation, as of the heart.

PALSY. Same as paralysis.

PAPILLA. The optic disk.

PARALYSIS. Loss of sensation or voluntary motion.

PARENCHYMA. The distinctive or functional elements of an organ in contradistinction to the sustentacular (sustaining, supporting) elements.

PARESIS. 1. Slight paralysis.

2. General paralysis of the insane.

PATHOGNOMONIC. Characteristic; peculiar to.

PATHOLOGY. The science of diseases.

PERIVASCULAR. Surrounding a vessel.

PERIVASCULITIS. Inflammation of the adventitia of vessel-walls.

PHLEBOSCLEROSIS. Hardening of the coats of a vein.

PHLYCTENA. A vesicle with serous contents; a blister.

PHOTOPHOBIA. A hyperesthetic sensitiveness to light.

PHYSIOLOGY. The science of the functions of the body.

PIGMENT. An organic coloring matter.

PLEXUS. A network of nerves or veins.

PNEUMONIA. Inflammation of the lungs.

PRODROME. A forerunner or sign of disease.

PROGNOSIS. Prediction of course and end of a disease.

PSYCHIC. Pertaining to the mind.

PTOMAIN. A crystallizable, nitrogenous basic substance, produced by bacteria in dead animal or vegetable matter.

PTOSIS. A drooping of the upper eyelid from paralysis. The term is also applied to an abnormal depression of other organs.

PULMONARY. Pertaining to the lungs.

PULSE. The expansile impulse of the arteries.

PUPIL. The round aperture in the iris of the eye.

PURULENT. Having the character of pus.

PUS. The fluid product of suppuration.

R

RECTUM. The lower part of the large intestine.

REFRACTION. The deviation of light on passing through mediums of different densities.

RENAL. Pertaining to the kidneys.

RETINITIS. Inflammation of the retina.

R., ALBUMINURIC, retinitis due to the presence of albumin in the urine.

R., HEMORRHAGIC, condition in which there are hemorrhages upon the retina.

R. PIGMENTOSA, retinal sclerosis with atrophy and pigmentation.

RHEUMATISM. A disease with fever, pain, inflammation, and swelling of the joints.

S

SARCOMA. A tumor of modified embryonic connective tissue.

SCARLATINA. An epidemic, exanthematous, contagious disease with fever and scarlet eruption.

SCLEROSIS. Induration and overgrowth of the connective tissue of an organ.

SCOTOMA. A dark spot in the visual field.

S., Scintillating, temporary amblyopia with subjective images, often an accompaniment of migraine.

SENILE. Pertaining to senility; aged.

SEPSIS. Putrefaction; septicemia.

SMALL-POX. A specific infectious disease with fever and papular eruption, followed by vesicles and pustules and the production of pits.

SPASM. A convulsive muscular contraction.

SPASMATIC. Pertaining to spasm.

SPECIFIC. 1. Peculiar, special. 2. Syphilitic. 3. A remedy of peculiar value.

SPHENOID. Cuneiform; wedge-shaped.

SPHINCTER. A muscle constricting an orifice.

SPINAL. Pertaining to the spine.

SPIROCHAETA PALLIDA. The germ of syphilis.

SPLEEN. An oval viscus behind the outer end of the stomach.

STASIS. Stagnation of the blood-current.

STENOSIS. A narrowing or constriction.

S., MITRAL, stenosis of the left auriculoventricular orifice.

STETHOSCOPE. A tube for conveying sounds in auscultation.

STRABISMUS. A condition in which the visual axes fail to meet at the objective point from incoordination of the eye-muscles; squint.

SUBJECTIVE. Internal; pertaining to one's self.

SUPRAORBITAL. Above the orbit.

SYMPTOM. A phenomenon of a disease usually subjective in character.

SYPHILIS. A chronic, infectious, venereal disease, which may also be hereditary, inducing cutaneous and other lesions; it is due to *Treponema pallidum* (also called *Spirochaeta pallida*).

SYSTEM. Methodic arrangement of parts.

S., SYMPATHETIC NERVOUS, a series of ganglions and nerves dominating the viscera and involuntary muscular system.

SYSTEMIC. Pertaining to a system or to the body as a whole.

SYSTOLE. The contraction of the heart and arteries.

T

TACHYCARDIA. Abnormal rapidity of cardiac action.

TACTILE. Pertaining to the sense of touch.

TEMPORAL. Pertaining to the temple.

TETANUS. A disease with spasmodic and continuous contraction of the muscles.

THERAPEUTICS. A branch of medical science concerned with the application of remedies and the treatment of the disease.

THORACIC. Pertaining to the chest.

THROMBOSIS. The formation of a thrombus.

THROMBUS. A blood-clot in a vessel at the point of obstruction.

THYROID. Scutiform; shield-shaped.

T. GLAND, a ductless glandular body at the upper part of the trachea, consisting of two (2) lateral lobes connected centrally by an isthmus.

TINNITUS. A ringing sound in the ear.

TONSIL. A glandular organ on each side of the fauces, or mouth and throat.

TOXEMIA. A poisoned state of the blood.

TOXIN. An amorphous, nitrogenous poison, formed by bacteria in both living tissues and dead substances.

- TRIGEMINUS. The fifth nerve; motor and sensory nerve to eye, face, tongue and teeth.
- TROCHLEAR. The fourth cranial nerve supplying the superior rectus muscle.
- TUBERCULOSIS. An infectious disease due to a specific bacillus, characterized by the formation of tubercles.
- TUMOR. 1. A swelling; an abnormal enlargement.
2. A new growth not the result of inflammation.
- TUNICA. An enveloping or lining membrane.
T. ADVENTITIA, the outer coat of an artery.
- TYMPANUM. The middle-ear cavity.
- TYPHOID. Resembling typhus.
T. FEVER, a continued, acute, infectious fever, with intestinal lesions, eruptions, etc.

U

- ULCER. Suppuration upon a free surface; an open sore.
- UNSTRIATED MUSCLE. Involuntary muscle fibers without transverse striations.
- UREA. The chief solid constituent of urine and principal nitrogenous product of tissue katabolism.
- UREMIA. The symptoms due to a toxic condition of the blood from accumulation of substances normally excreted by the kidneys.
- URINALYSIS. The analysis of urine.
- UVEAL. Pertaining to the uvea, which is the choroid, ciliary body, and iris, as a whole.

V

- VALVE. A fold across a canal obstructing passage in one direction.
- VARICOSE. Swollen; knotted.
- VARICOSITY. 1. A varix.
2. The state of being varicose.
- VASA VASORUM. The vessels supplying the arteries and veins with blood.
- VASCULAR. Pertaining to vessels.
- VASOCONSTRICTOR. 1. Producing constriction of vessels.
2. A nerve or drug causing constriction of vessels.

VENOUS. Pertaining to a vein.

VENTRICLE. A small belly-like cavity.

V. of HEART, LEFT, that upon the dorsal and left side of the heart, and which, through the aorta, forces the blood over the general system.

VERTIGO. Giddiness; dizziness.

VIRULENCE. Noxiousness; malignity.

VIRUS. 1. A morbid product.

2. A pathogenic microbe.

VITREOUS. Glass-like. The posterior and principal humor of the eye-ball.

W

WASSERMANN'S TEST. A serum-test used in the diagnosis of syphilis.

X

XANTHELASMA. A new growth of the skin, flat or slightly raised, and yellowish in color.

XEROSIS. Dryness.

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